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Life Insurance Medical Directors

of America

FIFTY-SIXTH ANNUAL MEETING

James R. Gudger, M. D.
Editor

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Transactions
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FIFTY-SIXTH ANNUAL MEETING

The Fifty-sixth Annual Meeting of The Association of Life Insurance Medical Directors of America was held at the Hotel Pennsylvania in New York City, on Thursday and Friday, October 23 and 24, 1947.

PRESIDENT JIMENIS — This is our 56th meeting and we can look back with admiration on the work of our predecessors and with some satisfaction on the contributions of our own generation. Altogether, a good deal has been added to our knowledge of medical underwriting. A nice balance has been established and maintained between clinical medicine brought to us by outstanding leaders in their specialties, and mortality studies by men engaged in insurance work and from insurance sources. This is the base that supports medical underwriting now. Only a short time ago clinical medicine was almost our only tool, and while we knew the short term effects of certain impairments, or histories of impairments, we did not know just how much importance should be attached to these, or for how long a period such histories constituted an insurance hazard. Mortality studies have provided an answer to some of our questions, and our decisions are now less vulnerable to argument and less likely to meet differing opinions.

The clinician, by means of more extensive and precise study and investigation, learns more about his individual cases, but he is rarely able to follow large numbers of patients

sufficiently long to measure their longevity. It is therefore not surprising that many heart murmurs are viewed optimistically by them, and yet we know how much more it costs to insure such lives. We still receive letters from family doctors minimizing dangers of small degrees of hypertension. This is not surprising because many such patients are lost sight of long before signs of myocardial failure or some vascular accident brings them to some other doctor. In a mortality study, however, the effects of such impairments are inescapable. As you know, each time we have studied blood pressure we have confirmed and strengthened our opinion that small degrees of hypertension produce a measurable increase in mortality, and that even transient hypertension is of some importance. Our opinion on this subject has been confirmed by Levy, White, Stroud and Hillman, who reported on the effects of elevated blood pressure, tachycardia and overweight on 22,741 Army officers. The final publication in this series appeared in the Journal of the American Medical Association on September 13, 1947, Vol. 135, page 77, under the title, "Sustained Hypertension".

Mortality studies began to appear at our meetings in 1907, and their value was immediately recognized. Some of you will remember Dr. Fisher, of the Northwestern Mutual, and his blood pressure studies which began in 1912. You will remember the varied list of mortality studies provided by Dr. Oscar Rogers and Mr. Arthur Hunter of the New York Life. The late Dr. Willard called these two investigators the medico-actuarial song and dance team. Well, their rhythm was catching and, recognizing the importance of such work, many other members of this Association produced similar studies. The Medico-Actuarial Committee produced the Medical Impairment Study of 1929, and it was followed by the Impairment Study of 1938, the Blood Pressure Study of 1939, and others. Not all of these studies carry with them the same degree of conviction. In reviewing the Medical Impairment Study and in spite of the larger number of cases, you will come across several categories subject to criticism. We have

to admit our handicaps and we are learning that it is useless to spend time and money building mortality tables which are not convincing when completed. When you decide to make a study, be sure you select something sufficiently factual. Do not depend on unsupported histories. A man might say, or might have been told, that he had pleurisy two years ago; but that proves nothing but a pain in the chest. However, if he mentioned empyema and your examination showed the scar of thoracotomy, you would have something credible. A man might have been told he had a duodenal ulcer but unless you had a statement from his physician, or he had been operated on, you would not know if it was in the duodenum or the stomach, or indeed if he had an ulcer.

If your study is built on a firm foundation, you should have no difficulty in upholding action in accordance with its results, regardless of what any other company might be doing at the same time.

The present day mortality study demands large numbers of cases presenting only the impairment to be studied and none other — they must be homogeneous as to grade, color and sex. Even when you find three or four thousand such cases in your files, by the time you separate them into the necessary categories you are likely to discover that the number of deaths in some of them is not convincing. The study itself takes a great deal of time and much clerical help. It follows that all companies are not always in position to undertake such work.

At the Medical Section of the American Life Convention meeting earlier this year, Dr. Bonnett mentioned a hope that our actuaries would devise some quicker and easier check which, although not conclusive, would at least point out a trend in mortality and give us some idea of the results to be expected. I would like to repeat this hope. Clinicians hardly ever use mortality tables in calculating results. Have we been thinking too strictly about this? You might ask the same question of your own actuaries, for there are still

too many conditions about which we need more information. Do not wait for another medical impairment study. Study what you have and report it. Perhaps the following year some other company will be able to compare their results with yours. Nor should you wait to be invited to present the results of your work. Tell the President or the Secretary, or the Chairman of the Program Committee what you have to offer — he will probably be delighted to have you take part in the program.

Thus, combining what we learn from clinicians with our own studies, we will continue to make progress in the future as we have in the past.

Our first paper, gentlemen, is "Mortality Study of Applicants for Insurance Given a Glucose Tolerance Test." It is one that we have been planning in the Metropolitan Life for many years, and our good friend, Dr. Rexford W. Finegan, is going to tell you all about it. Dr. Finegan!

MORTALITY STUDY OF APPLICANTS FOR INSURANCE GIVEN A GLUCOSE TOLERANCE TEST

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Three diseases are receiving more attention from Medical Directors than any others. These are tuberculosis, heart disease, and diabetes. Happily, tuberculosis is on the wane; heart disease continues to take up a good deal of our time, and now for some years our interest in diabetes has been growing. At first we sought only to avoid those who had symptoms or signs of diabetes, but lately some intelligently courageous companies have been underwriting diabetics. The special tests that we have been using are urine and blood analyses.

For this purpose the limitations of the urine test have long been known to all of us. The routine of life insurance medical examination does not permit the taking of successive samples of urine over a period of time, as would be required for the

establishment of a clinical diagnosis, and even so, in the absence of symptoms the diagnosis cannot be made without the blood sugar determinations. Moreover, with the advent of insulin diabetics with a good knowledge of how to manage their disease could and did sometimes control their glycosuria for an insurance examination. While it is true that Lewis and Benedict's (1) picrate method of estimating the amount of sugar in the blood was published in 1915, and studies of the effect of glucose and other carbohydrates on blood sugar levels (2, 3) had been made abroad even earlier, knowledge of carbohydrate metabolism was not far advanced, and standards for the diagnosis of diabetes by the new methods had not yet been established. In 1919 Folin and Wu (4) published a new system of blood analysis including the determination of blood sugar. The simplicity of their new system greatly facilitated the performance of blood sugar tests and consequently they were done in increasing numbers. The discovery of insulin gave great impetus to the use of these new tests. Folin and Malmros (5, 6) published their micro method for the determination of blood sugar in 1928, and it was soon realized that this method made it possible to obtain blood samples which could be sent to a central laboratory by post.

The Metropolitan (7, 8) developed a method for the collection and preservation of blood in capillary tubes for mailing to the Home Office in 1933. Only technical difficulties in obtaining satisfactory specimens and time consumed by the test have limited its applicability.

Our first glucose tolerance test was done at the Home Office in 1927. At the beginning we did only a few, and it was not until late in 1931 that they were given in any great numbers. Since October, 1931, the test has been offered to approximately 17,000 applicants of whom about two-thirds took it. About 10,000 good collections were obtained and

about 5,000 persons refused the test. Eighty-nine per cent of the field tests were satisfactorily collected. In comparison with the total number of persons medically examined during the period 1932–1946, the 17,000 offers of the test represented a little over 2 per 1,000 medically examined and the number of completed satisfactory cases amounted to about 1.2 per 1,000.

Rules for Ordering Glucose Tolerance Tests

A. Prior to 1932 there were no definite rules for ordering the test.

B. Between 1932 and 1935 a tolerance test was ordered if there was (1) 0.4 per cent sugar in two or more specimens, or (2) 0.5 or more per cent in one specimen.

C. From 1935 on more consideration was given to history and overweight when ordering the test.

D. Since mid-1942 the tolerance test has been given also when there is a history of two or more diabetics in the family.

Technique of Test

Prior to 1939

The test was begun either in the fasting state or at least 2½ hours after a meal. Fifty grams of glucose were given in a glassful of water. The samples of blood were collected in capillary tubes before the ingestion of glucose, and ½ hour and 2 hours afterward. A specimen of urine was collected at the beginning of the test and the bladder emptied. At the end of 2 hours, another specimen was obtained.

1939 and Since

Since 1939 it has been required that at least 4 hours elapse after the last meal. All urine voided during the test period is measured and a portion sent to the Home Office.

Laboratory Procedures

Prior to 1939

The blood was collected in accurately calibrated 0.1 ml. pipettes. Upon receipt of the samples in the laboratory, the blood proteins were precipitated with dilute tungstic acid and the sugar in the filtrates was determined by the method of Folin and Malmros (5). Urine sugar was determined by the Benedict picrate method (9).

1939 and Since

The blood samples were transferred in the laboratory to accurate 0.1 ml. pipettes. The sugar was estimated as described above. The method for urine sugar described by Folin and Svedberg (10) was used. This method removes non-sugar reducing substances by treatment with Lloyd's reagent and permutit. The sugar remaining in the treated filtrates is determined by the method of Folin (11–12). Unpublished results from our Biochemical Laboratory show that the picrate and the Folin methods are in substantial agreement when there is 0.4 per cent or more sugar. Values under 0.4 per cent by the picrate method actually correspond to considerably less true sugar.

General Considerations

Valuable as the glucose tolerance test is, we think that none of us would consider a single test carried out under conditions that are not ideal as the final answer to the state of the carbohydrate metabolism. In a sense, however, our problem is simplified when we stop short of making a diagnosis of the presence or absence of diabetes and confine ourselves to an underwriting opinion. There are other reasons why we should be thus cautious. The glucose tolerance test repeated on the same individual may vary considerably under different con-

ditions. Some of these are previous diet, emotion, pregnancy, endocrine disorders, liver disease, and in addition, insulin and infections will alter the response to the test. Any of these conditions may be present without showing any obvious signs. There are still differences of opinion among leaders in the field of diabetes as to the limits of the blood sugar values which may be considered diagnostic of the disease. Furthermore, while in non-diabetics we consider that the blood sugar should return to the fasting level in two hours, we do recognize that in some individuals with slow absorption of sugar, the return may be delayed.

We have used capillary arterial blood from the fingertip because this avoids venipuncture. The fasting values yielded by capillary and venous bloods are essentially similar but the peak value for capillary blood is considerably higher. The average difference is about 30 mg.

Description of Material

The present experience relates primarily to 5,532 applicants for Ordinary insurance, both males and females, from whom we received blood samples satisfactory for analysis and who, except for those with abnormal urine and blood sugar findings, were eligible for standard insurance. The study includes cases from 1927 to 1944, whether accepted for insurance, either standard or substandard, or rejected.*

Thus, we have eliminated all those rated as Substandard because of occupation or other medical impairments. From this latter group, however, we segregated those rated for overweight but otherwise Standard (except, of course, with respect to blood sugar levels and glycosuria in some in-

* Some of the cases declined prior to 1937 were lost for use in this study because the application papers were destroyed under the Company's program of records destruction. However, some of them had been systematically followed for some time and the experience on them is included since 1937, since which time records on all declined applications were available.

stances), and the results on these cases, numbering 374, are reported on separately in this paper. Those cases which were rated substandard for other urinary impairments were also segregated for study, but their number was found to be too small to warrant tabulation.

The cases were traced to the anniversary in 1946 of the date of policy issue or of the date of examination in cases when no policy was issued. We have been thorough in our tracing and have used a whole battery of methods including, as you know, the records of other insurance companies for whose assistance we are extremely grateful. We were able to trace about three-fourths of the cases through insurance in our own or other companies. To trace the remainder, we used every clue we had or could find. Thus, we made inquiry through our District Offices, if the Agent who wrote the original application was still in the Company's service or if insurance on a relative of the applicant was known to be in force. We wrote to applicants and to physicians. We used many other sources, such as telephone, professional and business directories, license records—automobile drivers' licenses, motor vehicle registration, pharmacists and other occupations where licenses were required—labor unions, and employers. A credit agency helped on those cases that were troublesome or elusive or when circumstances made it advisable not to use other approaches. In order to trace some individuals, we had to use several methods.

Altogether, we have traced more than 98 per cent of the cases, thus losing less than 2 per cent, and for some of the latter we had information covering several years. We consider this a good record in view of the comparatively long duration of the experience and the considerable population movements that have taken place during the period of the study. The average duration of observations in this group was seven and one-third years.

We have evaluated the experience on these cases against the contemporaneous mortality experience among medically examined standard risks in our Ordinary Department. Because the vast majority of the cases were examined since the latter part of 1931, the standard table used for evaluation relates to the years from 1932. Mortality values for the durations of insurance beyond those included in our standard table were obtained by a simple graphic extension of the observed rates of the table.

In our analysis we have limited ourselves pretty much to the findings on ½ hour and 2 hour samples, principally because we are not certain that all of our initial specimens were taken as directed. Apart from that, we found generally that the initial blood sugar was high only when the ½ hour and 2 hour values were high; and conversely, there were relatively few cases with high initial values and normal subsequent levels. We considered the tabulation of the latter group of cases, but the number of deaths in this category was too small to make it worth while. In view of the tendency for both the ½ hour and 2 hour values to conform either to a normal or to an abnormal pattern, the number of cases with borderline values or with distinct peculiarities at the ½ hour level was relatively few.

Mortality Analysis

The first and major portion of our analysis relates to the grouping of the data on the basis of the ½ hour and 2 hour blood sugar regardless of the presence or absence of glycosuria. Our data are most abundant for those cases with ½ hour blood sugar of 180 mg. per 100 c.c. or less, and 2 hour blood sugar of 110 or less. The samples in detailed classes by successive ½ hour and 2 hour readings were too small to yield reliable results. For this reason, it was necessary to make

some rather broad groupings. Relatively few cases had ½ hour blood sugar over 200, unless the 2 hour value was high. The ½ hour blood sugar was under 160 in most cases with 2 hour levels under 120.

The ratios of actual to expected deaths follow a pattern that is familiar to those accustomed to mortality studies in that they do not show an entirely smooth progression, with increasing blood sugar levels. We must constantly keep in mind, however, that the credibility of the results is in direct proportion to the size of the group as measured by the exposure or the number of deaths.

As Table I shows, our experience with all cases in which the ½ hour blood sugar was 200 or less and the 2 hour blood sugar 110 or less has been consistently favorable, all of the groups yielding an essentially normal mortality. The cases with ½ hour blood sugar of 200 or less and 2 hour blood sugar between 111 and 120 showed a mortality above normal, and in the aggregate not much different from the cases with ½ hour blood sugar of 200 or less and 2 hour blood sugar between 121 and 140. While none of the groups is especially large, we do not think that these results should be dismissed lightly. The small experience with cases having ½ hour values of 200 or less and 2 hour values between 141 and 160 has been favorable, but the mortality of those having ½ hour sugar of 200 or less with 2 hour values of 161 and over has been well above normal.

Another set of combinations was made consisting of two groups of cases having 2 hour blood sugar between 121 and 140, namely, those with ½ hour levels of 160 or less and of 161 to 180. Both these groups experienced a mortality of 155 per cent, with the ratio for the latter based upon only 10 deaths. (Table 1A)

Table I

CASES CLASSIFIED ACCORDING TO ½ HOUR AND 2 HOUR BLOOD SUGAR LEVELS

Table I

CASES CLASSIFIED ACCORDING TO ½ HOUR AND 2 HOUR BLOOD SUGAR LEVELS

Percent Actual of Expected Deaths by Contemporaneous Mortality Experience on Standard Risks
Metropolitan Life Insurance Co., Ordinary Dept., 1927 to 1944, Traced to Anniversary in 1946.

Age at Issue; Duration; ½ Hr. Blood Sugar (mg. %)	2 Hour Blood Sugar (mg. %)																	
	110 or Less			111–120			121–130			131–140			141–160			161 and Over		
	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected
All Ages; All Durations																		
160 or less	17,703	73	104	2,303	18	149	2,307	19	142	1,748	16	145	2,168	11	81	1,456	14	153
161 to 180	6,343	29	99	1,343	10	126												
181 to 200	3,189	17	100															
201 and over				933	5	81				308	9	356	317	4	*	1,264	21	235
Under age 40																		
160 or less	11,817	22	95	1,299	2	*	1,152	2	*	670	1	*	994	2	*	492	1	*
161 to 180	3,640	9	111	587	–	–												
181 to 200	1,560	2	*															
201 and over				372	1	*				89	2	*	91	1	*	249	–	–
Ages 40–49																		
160 or less	4,407	29	110	704	9	203	830	9	162	782	10	193	764	3	*	583	7	233
161 to 180	2,002	9	76	541	5	145												
181 to 200	1,173	7	97															
201 and over				347	1	*				143	4	*	155	2	*	483	5	204
Ages 50 and over																		
160 or less	1,479	22	108	300	7	140	325	8	150	296	5	115	410	6	95	381	6	117
161 to 180	701	11	119	215	5	155												
181 to 200	456	8	129															
201 and over				214	3	*				76	3	*	71	1	*	532	16	268
Durations, 1 to 5 yrs.																		
160 or less	10,182	27	93	1,305	10	219	1,226	7	145	935	7	175	1,157	6	119	1,105	9	160
161 to 180	3,763	15	118	769	1	*												
181 to 200	1,845	6	85															
201 and over				506	2	*				149	2	*	174	–	–	985	14	230
Durations, 6 yrs. and over																		
160 or less	7,521	46	112	998	8	107	1,081	12	140	813	9	127	1,011	5	58	351	5	142
161 to 180	2,580	14	85	574	9	181												
181 to 200	1,344	11	110															
201 and over				427	3	*				159	7	395	143	4	*	279	7	246

Table 1A

Cases with ½ Hour Blood Sugar Levels of 160 mg. % or Less and of 161–180 mg. % with 2 Hour Blood Sugar of 121–140 mg. %

Age at Issue; Duration; ½ Hr. Blood Sugar (mg. %)	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected
All Ages; All Durations			
160 or less	2,253	21	155
161 to 180	1,109	10	155
Under Age 40			
160 or less	1,101	2	*
161 to 180	455	1	*
Ages 40 - 49			
160 or less	820	12	224
161 to 180	493	6	184
Ages 50 and over			
160 or less	332	7	123
161 to 180	161	3	*
Durations, 1 to 5 yrs.			
160 or less	1,168	7	150
161 to 180	647	5	192
Durations, 6 yrs. and over			
160 or less	1,085	14	158
161 to 180	462	5	131

* Less than 5 deaths

All the groups with ½ hour values over 200 yielded high mortality ratios except the one with 2 hour values of 120 or less, a small group with only 5 deaths. The largest group in this experience consisted of those with 2 hour blood sugar exceeding 160, for which the ratio is nearly 2½ times the expected.

There are sizable variations in the ratios with age and with duration of insurance, but for the most part these do not show any consistent pattern.

There is interest, of course, in the mortality among those cases with blood sugar well above the normal level, and we have analyzed, therefore, the experience among those cases with 2 hour blood sugar exceeding 200, and for comparison those having 2 hour sugar between 161 and 200. For those over 200, we found a mortality ratio of 242 per cent. This is appreciably higher than for cases with 2 hour sugar between 161 and 200. The subdivisions of this table may be of interest, although the small number of deaths robs these categories of much significance.

Table II

CASES WITH 2 HOUR BLOOD SUGAR LEVELS OF 161–200 mg. % AND OF 201 mg. % OR OVER

Percent Actual of Expected Deaths by Contemporaneous Mortality Experience on Standard Risks, Metropolitan Life Insurance Co., Ordinary Dept., 1927 to 1944, Traced to Anniversary in 1946.

Age at Issue; Duration; Blood Sugar Level	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected
All Ages; All Durations			
161 – 200	1532	16	157
201 and over	1188	19	242
Under Age 40			
161 – 200	450	1	*
201 and over	291	–	–
Ages 40 – 49			
161 – 200	587	8	267
201 and over	479	4	*
Ages 50 and over			
161 – 200	495	7	112
201 and over	418	15	310
Durations 1 to 5 yrs.			
161 – 200	1150	9	146
201 and over	940	14	253
Durations 6 yrs. and over			
161 – 200	382	7	173
201 and over	248	5	215

* Less than 5 deaths.

The only cases that we have analyzed with respect to the initial blood sugar are those having 2 hour readings exceeding 140. Three groups were distinguished, namely, those with initial sugar of 120 or less, those between 121 and 140, and those over 140. We found that for each of these three groups the mortality ratios were above normal and progressively so with increasing height of the initial level.

Table III

CASES WITH 2 HOUR BLOOD SUGAR LEVEL OF 141 mg. % OR MORE, CLASSIFIED ACCORDING TO THE INITIAL LEVEL

Percent Actual of Expected Deaths by Contemporaneous Mortality Experience on Standard Risks, Metropolitan Life Insurance Co., Ordinary Dept., 1927 to 1944, Traced to Anniversary in 1946.

Age at Issue; Duration; Initial Blood Sugar	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected
All Ages; All Durations			
120 or less	2657	21	126
121 – 140	904	10	168
141 and over	1295	19	209
Under Age 40			
120 or less	1132	3	*
121 – 140	285	–	–
141 and over	315	1	*
Ages 40 – 49			
120 or less	935	9	158
121 – 140	388	5	211
141 and over	488	3	*
Ages 50 and over			
120 or less	590	9	105
121 – 140	231	5	174
141 and over	492	15	259
Durations 1 to 5 yrs.			
120 or less	1642	13	175
121 – 140	604	4	*
141 and over	977	12	204
Durations 6 yrs. and over			
120 or less	1015	8	86
121 – 140	300	6	233
141 and over	318	7	220

* Less than 5 deaths.

Summation of Experience in Three Broad Blood Sugar Groups

When the experience in the detailed groups is aggregated into three broad blood sugar groups — normal, borderline, and high — based upon the relationship of the ½ hour and 2 hour levels (as shown in Table IV), the results seem to give a well ordered pattern except in a few instances — those with relatively small numbers of deaths.

Table IV

Class	Blood Sugar (mg. per cent)	
	½ hour	2 hour
Normal	200 or less	120 or less
Borderline	201 or more	120 or less
	200 or less	121 to 140
High	201 or more	121 to 140
	Any amount	141 or over

Table V shows that the normal group in the aggregate has a mortality ratio of 108 per cent, the borderline group 131 per cent, and the high group, 162 per cent. When the data are considered according to ages at issue, and by duration, the mortality in the normal group is the lowest in every instance, except at ages under 40; but here the groups are too small to be significant. In every instance, the mortality of the high group is the worst. In a few instances, however, the differences are smaller than one would expect. At age 50 and over, the mortality ratio of the normal cases is quite high, 120 per cent, and not much below the mortality in the borderline cases. At ages 40 to 49, the mortality in the borderline group is rather close to the mortality in the high group.

Table V

CASES CLASSIFIED INTO THREE BROAD BLOOD SUGAR GROUPS

Percent Actual of Expected Deaths by Contemporaneous Mortality Experience on Standard Risks, Metropolitan Life Insurance Co., Ordinary Dept., 1927 to 1944, Traced to Anniversary in 1946.

Age at Issue; Duration; Blood Sugar Group †	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected
All Ages; All Durations			
Normal	30,881	147	108
Borderline	4,988	40	131
High	5,513	59	162
Ages under 40			
Normal	18,903	35	90
Borderline	2,194	4	*
High	1,915	6	138
Ages 40 – 49			
Normal	8,827	59	110
Borderline	1,959	20	153
High	2,128	21	167
Ages 50 and over			
Normal	3,151	53	120
Borderline	835	16	128
High	1,470	32	164
Durations 1 – 5 yrs.			
Normal	17,864	59	105
Borderline	2,667	16	144
High	3,570	31	168
Durations 6 yrs. and over			
Normal	13,017	88	110
Borderline	2,321	24	123
High	1,943	28	155

† For definition see Table IV.

* Less than 5 deaths.

The number of cases at ages under 30 (figures not shown) is comparatively small, and a separate study of this group shows exceptionally good results in the normal group; but, unfortunately, in the other groups the experience is too small to throw much light on the matter. It is true, however, that in the borderline group in an experience of 648 years of life there were no deaths, and in the high group, with 486 years of life, only one death occurred.

Mortality in Relation to Presence or Absence of Glycosuria and According to Build

For this part of the analysis, we have used the three broad blood sugar categories—normal, borderline, and high—as defined earlier. Three divisions were made according to the amount of sugar in the 2 hour urine specimen—negligible (less than 0.4 per cent), moderate (0.4 per cent to 1 per cent), and 1 per cent and over. As for build, the groups were divided into those less than 10 per cent above average weight and those 10 per cent or more overweight up to the limit allowed for standard insurance in our underwriting practice. To simplify matters, we shall refer to these as the average weight and moderately overweight groups respectively.

This analysis indicates that the amount of sugar in the urine 2 hours after glucose did not significantly affect the results, although the figures must be interpreted with caution because of the small size of some of the groups. The major factors appear to be the level of the blood sugar and overweight. For example, among average weight cases with normal blood sugar, the mortality ratio was 99 per cent for those with negligible glycosuria; 125 per cent for those with moderate glycosuria; and 106 per cent with glycosuria of 1 per cent or more; and among those moderately overweight, the ratios were 135 per cent, 114 per cent, and 66 per cent respectively, the last ratio being based on only 4 deaths.

Table VI

NORMAL, BORDERLINE AND HIGH BLOOD SUGAR CASES, CLASSIFIED ACCORDING TO AMOUNT OF GLYCOSURIA IN 2 HOUR SPECIMEN AND ACCORDING TO WEIGHT GROUP

Percent Actual of Expected Deaths by Contemporaneous Mortality Experience on Standard Risks, Metropolitan Life Insurance Co.. Ordinary Dept., 1927 to 1944, Traced to Anniversary in 1946.

Blood Sugar Group †; Build	Amount of Glycosuria								
	Less than 0.4%			0.4% but less than 1%			1% or over		
	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected
Normal									
Average weight	15,888	68	99	3,907	23	125	3,301	17	106
Moderately overweight	4,860	27	135	1,391	7	114	1,217	4	*
Borderline									
Average weight	1,507	13	137	854	5	93	950	2	*
Moderately overweight	721	10	233	415	5	200	497	5	187
High									
Average weight	1,017	11	152	777	4	*	1,705.	20	169
Moderately overweight	421	4	*	345	2	*	1,223	17	221

† For definition see Table IV.

* Less than 5 deaths.

The relative unimportance of glycosuria in our study is further brought out in the next table which shows the results by blood sugar groupings according to the amount of glycosuria regardless of weight. In both the normal and borderline blood sugar groups, the lowest mortality ratios are found among cases with glycosuria of 1 per cent or over. Only in the high blood sugar group is the ratio highest for those with glycosuria of 1 per cent or over, but in this group the ratio for those with negligible glycosuria is also high.

In contrast are the results by blood sugar groupings according to weight, regardless of the amount of glycosuria. In each blood sugar group, the mortality of those 10 per cent or more overweight is higher than that of applicants less than 10 per cent overweight.

The cases since 1939 have been analyzed according to the amount of sugar excreted in the 2 hour test period. Two groups were studied, namely 60 mg. to 1 gm., and 1 gm. or over. The numbers of cases when classified by blood sugar levels were too small to yield significant results, but the mortality ratios seem to indicate that the quantity of sugar excreted was not important. It may be that our examiners in many cases failed to obtain accurate figures on the total volume of urine voided in the 2 hours.

Cases Rated Because of Overweight

Among the 374 cases in this group, there were 22 deaths. Again, when subdivided according to the three broad blood sugar groups, the experience proved to be rather thin and not very informative. All the groups showed mortality ratios exceeding 100 per cent. The ratio was highest, nearly twice the expected, in those with normal blood sugar.

Mortality Results According to Rating

The Metropolitan writes substandard insurance, and its method of handling these risks is to place them in classes according to the expected extra mortality. For this purpose,

Table VII

NORMAL, BORDERLINE AND HIGH BLOOD SUGAR CASES, CLASSIFIED ACCORDING TO AMOUNT OF GLYCOSURIA IN 2 HOUR SPECIMEN.

Percent Actual of Expected Deaths by Contemporaneous Mortality Experience on Standard Risks, Metropolitan Life Insurance Co., Ordinary Dept., 1927 to 1944, Traced to Anniversary in 1946.

Blood Sugar Group*	Amount of Glycosuria								
	Less than 0.4%			0.4% but less than 1%			1% or over		
	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected
Normal	20,748	95	108	5,298	30	122	4,518	21	95
Borderline	2,228	23	166	1,269	10	127	1,447	7	80
High	1,438	15	152	1,122	6	87	2,928	37	189

* For definition see Table IV.

Table VIII

NORMAL, BORDERLINE AND HIGH BLOOD SUGAR CASES, CLASSIFIED ACCORDING TO WEIGHT GROUP

Percent Actual of Expected Deaths by Contemporaneous Mortality Experience on Standard Risks, Metropolitan Life Insurance Co., Ordinary Dept., 1927 to 1944, Traced to Anniversary in 1946.

Blood Sugar Group*	Average Weight			Moderately Overweight		
	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected
Normal	23.096	108	105	7,468	38	118
Borderline	3,311	20	95	1,633	20	211
High	3,499	35	146	1,989	23	187

* For definition see Table IV.

there are three substandard classes, namely Intermediate, Special Class, and Special Class B. They allow a range of extra mortality from 30 per cent to a maximum of 175 per cent. Risk groups with an expected mortality above this limit are declined.

Prior to 1939, we were strict in underwriting anyone who originally showed glycosuria, even though later the glucose tolerance test was shown to be normal. At that time we considered 200 mg. per 100 cc. of blood as the limit of normal at the end of ½ hour, and 110 as the limit of normal at the end of 2 hours. We declined all who had over 120 at the end of 2 hours. In 1939, these normal limits were raised to 220 at ½ hour and 120 at 2 hours. Those having more than 130 at 2 hours were rejected. Since 1939, as a result of these somewhat more liberal standards, slightly more than half of the otherwise unimpaired cases included in this study were granted standard insurance. The effect of this liberalization was to reduce greatly the proportion of those limited to substandard insurance. The proportion declined for insurance has been about the same before and since 1939.

In the analysis of the results according to the ratings, we have tabulated the data according to the two periods before and since 1939, as well as for the aggregate of the two periods. In comparing the ratios for the two periods, however, it should be borne in mind that we have used the same standard mortality table to measure both parts of the experience, and since the period of this investigation, 1932–1946, is characterized by a steadily declining mortality, this standard tends to be somewhat too severe for the earlier cases and too lenient for the later cases. If adjustment were made for this, the margin between the earlier and later periods with respect to the mortality ratios would be appreciably smaller. In the later period, the average exposure is rather short, and two of the classes are too small to yield significant comparisons with the earlier period.

The aggregate standard experience yielded a ratio of 112 per cent actual of expected deaths. The experience on Intermediate risks showed a ratio of only 79 per cent, and Special Class and Special Class B combined (there being too few of the latter to segregate) showed a ratio of only 125 per cent. The mortality ratio for declined applicants was 155 per cent, or just about 1½ times the normal. In general, the results have been uniformly good at ages under 40. For ages 40 to 49, our Ordinary experience was slightly above the expected but within our limits. At age 50 and over, the Ordinary experience was somewhat unsatisfactory. There appear to be no essential differences according to duration of insurance. Our underwriting of the Ordinary and Intermediate cases was not too brilliant, and we were quite conservative on those issued Special Class and on those declined.

Among cases accepted for insurance, the mortality ratios for the more recent calendar years of issue are lower, with fair consistency by age at issue and duration, than for earlier cases. As already indicated, this is in part to be expected and is explained by the general downward trend in mortality. Among declined risks, however, the mortality ratios for early and for recent cases are about the same—157 per cent and 152 per cent respectively. This means, of course, that relatively the experience on the later declined cases is worse. This is brought out by comparing these ratios with those on cases accepted for Ordinary insurance. Among cases examined prior to 1939, the ratio for the declined was only about one-fourth higher than for those accepted for Ordinary; whereas among cases of 1939 and later it was nearly double that for the standard group.

Causes of Death

We have kept a systematic record of the causes of death reported in each of the various classes. We were particularly interested in the mortality from diabetes, but in view of the other conditions which are known to influence the blood

Table IX

CASES CLASSIFIED ACCORDING TO THE RATING GIVEN*

Percent Actual of Expected Deaths by Contemporaneous Mortality Experience on Standard Risks, Metropolitan Life Insurance Co., Ordinary Dept., 1927 to 1944, Traced to Anniversary in 1946.

Age at Issue; Duration; Rating	Date of Issue or Action								
	All Cases			1927–1938			1939–1944		
	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected	Years of Life Exposed to Risk	Actual Deaths	Percent Actual of Expected
All Ages, All Durations									
Ordinary	20,307	92	112	13,576	75	123	6,731	17	79
Intermediate	7,403	28	79	5,609	23	79	1,794	5	80
Special Class	2,692	17	125	2,322	17	139	370	–	–
Declined	10,223	104	155	6,895	79	157	3,328	25	152
Ages under 40									
Ordinary	12,960	24	93	8,775	21	110	4,185	3	**
Intermediate	4,097	6	71	3,047	5	75	1,050	1	**
Special Class	1,492	2	**	1,300	2	**	192	–	–
Declined	4,085	11	115	2,879	8	106	1,206	3	**
Ages 40 to 49									
Ordinary	5,519	36	111	3,576	28	118	1,943	8	92
Intermediate	2,439	12	80	1,885	11	90	554	1	**
Special Class	820	8	157	686	8	178	134	–	–
Declined	3,879	41	164	2,563	33	175	1,316	8	131
Ages 50 and over									
Ordinary	1,828	32	133	1,225	26	144	603	6	99
Intermediate	867	10	82	677	7	68	190	3	**
Special Class	380	7	135	336	7	148	44	–	–
Declined	2,259	52	161	1,453	38	158	806	14	168
Durations, 1 to 5 yrs.									
Ordinary	12,387	37	100	6,493	24	121	5,894	13	75
Intermediate	4,232	13	91	2,693	8	86	1,539	5	100
Special Class	1,439	9	176	1,120	9	223	319	–	–
Declined	5,660	45	165	2,717	22	161	2,943	23	168
Durations, 6 yrs. and over									
Ordinary	7,920	55	122	7,083	51	124	837	4	**
Intermediate	3,171	15	71	2,916	15	75	255	–	–
Special Class	1,253	8	95	1,202	8	98	51	–	–
Declined	4,563	59	149	4,178	57	155	385	2	**

* Cases are included in accordance with the offer made, whether or not it was accepted. Cases are excluded from this tabulation if the offer was not clearly defined.

** Less than 5 deaths.

sugar level after glucose administration, we looked for the occurrence of liver disorders either as a primary or secondary cause of death, and occurrence of malignancies, especially those involving the liver and pancreas. We segregated also the deaths from coronary artery disease because we had long ago observed what seemed to be an inordinately high proportion of deaths from that cause among our cases.

While the facts on causes of death are of distinct interest, it is obvious that in most of the groups the numbers are too small to show up significant deviations from normal, and, in fact, nothing like a clear picture appears except for the three broad classes of blood sugar—normal, borderline, and high. For these we have made some estimates of the deviations on the basis of the findings in the normal group, with due consideration for differences in the age distribution of the cases at issue.

The first fact of importance is that while the mortality from diabetes is high in the borderline and high blood sugar groups, this disease is not by any means the first cause of death in any of the groups, nor does it account for all the extra mortality observed in these groups. Altogether, there were only 19 cases in which diabetes was reported as the primary cause of death out of the entire number of 246 deaths in the main part of the experience, and 6 deaths in which the disease was a contributory cause. Taking the normal group as standard, there would be expected only about one death from diabetes in the borderline group as against 4 reported, and between 1 and 2 expected in the high group as against 11 observed. No deaths from the disease have been recorded among the applicants under age 40 at issue. The mortality from cardiovascular disease was generally high in the borderline and high blood sugar groups—about one and one-half times that in the normal group. There were too few deaths under age 40 in the borderline and high groups to yield any significant differences on this score, but relatively the greatest excess mortality from cardiovascular disease was observed among

those between ages 40 and 49 at issue or examination, and the relative mortality from this cause was as high in the borderline group as in the high blood sugar group. The excess mortality from coronary artery disease was about the same as that for the entire cardiovascular group.

There were only 3 deaths from chronic nephritis, two of which occurred in the normal group, so that there is consequently no indication of excess kidney disease among these cases. No information was developed which might indicate that intercapillary glomerulosclerosis is an important cause of death. The mortality from cancer was greatest in the high blood sugar group, with 11 deaths as against only 6 expected. The numbers of deaths from other causes were too small to show any special characteristics. Consequently, we cannot demonstrate any relationship between high blood sugar and mortality from liver disease.

Among cases with 2 hour blood sugar over 200 mg. per 100 cc. of blood, six of the 19 deaths were recorded as due to diabetes, and one other case was diabetic but died of cancer of the cecum. Eight of the deaths in this group were from cardiovascular diseases—five of them coronary.

Examination of the causes of death in the groups classified according to the amount of sugar in the urine at the end of the 2 hour test period showed that there was no particular relationship between the occurrence of diabetes and glycosuria in the normal and borderline blood sugar groups. As was to be expected, in the high blood sugar group the diabetes cases were most frequent among those with marked glycosuria.

The proportion of deaths from diabetes was slightly greater among those who were moderately overweight than among those of average weight or less, and it is noteworthy that most of the diabetes deaths in the latter group were found among cases with high blood sugar and marked glycosuria. One cannot overlook the likelihood that some of these may have been true diabetics who had lost weight although this was not admitted in the history.

Table X

NUMBER OF DEATHS FROM SPECIFIED CAUSES IN NORMAL, BORDERLINE AND HIGH BLOOD SUGAR GROUPS AND RELATIVE FREQUENCY OF THE CHIEF CAUSES IN THE BORDERLINE AND HIGH BLOOD SUGAR GROUPS, WITH THE NORMAL GROUP TAKEN AS STANDARD.

Metropolitan Life Insurance Co., Ordinary Dept., 1927 to 1944, Traced to Anniversary in 1946.

(Relative frequency of chief causes denoted by following symbols: + high; ++ very high. Where no symbols appear, number of deaths was too few to be of any significance.)

Cause of Death	Number of Deaths		
	Normal	Borderline	High
Total	147	40	59
Diabetes, Total	5	4++	16++
Primary	4	4++	11++
With Coronary Artery Disease	1	1	5++
Secondary	1	–	5++
Cardiovascular, Total	58	21+	26+
Coronary Artery Disease	40	15+	16+
Other Chronic Heart Disease	12	1	5+
Cerebral Hemorrhage, etc.	4	2	3
Other	2	3	2
Chronic Nephritis	2	–	1
Cancer	24	7	11+
Liver and Biliary Tract Disease	4	–	1
Pneumonia and Influenza	11	3	1
Accidents and Suicide	21	3	2
Other	23	2	6

When the causes of death are examined according to the action taken, it is a source of satisfaction to know that only one of the deaths reported from diabetes occurred among those granted standard insurance and two of the diabetes

deaths were in the substandard group. The remaining 16 deaths and all of those associated with coronary disease were in the declined group.

Table XI

CHIEF CAUSES OF DEATH AMONG APPLICANTS TAKING GLUCOSE TOLERANCE TEST CLASSIFIED ACCORDING TO RATING GIVEN*

Metropolitan Life Insurance Co., Ordinary Dept., 1927 to 1944, Traced to Anniversary in 1946.

Cause of Death	Standard	Substandard	Decline
Total	92	45	104
Diabetes, total	2	2	21
Primary	1	2	16
with coronary artery disease	–	–	7
Secondary	1	–	5
Cardiovascular, total	37	18	48
Coronary artery disease	27	14	29
Other chronic heart disease	6	2	10
Cerebral hemorrhage, etc.	2	2	4
Other	2	–	5
Chronic nephritis	2	–	1
Cancer, total	15	7	19
Biliary tract	2	1	–
Liver and biliary tract disease	2	2	1
Gastric and duodenal ulcer	2	2	1
Pneumonia and influenza	8	3	4
Tuberculosis	–	–	–
Accidents	9	4	4
Suicide	4	1	2
Others	12	6	8

* Cases included in accordance with offer made, whether or not it was accepted. Cases are excluded from this tabulation if the offer was not clearly defined.

DISCUSSION

With due regard to the limitations of the glucose tolerance test as performed in connection with examinations for life insurance, the results of this mortality study are of real significance and raise some questions which deserve serious consideration.

First of all, our study indicates that we can usually judge the results of glucose tolerance tests on the basis of the blood sugar findings and disregard a moderate degree of glycosuria during the test. Our analysis also seems to indicate that while the ½ hour peak is of interest and may guide us in some cases, the blood sugar levels at the end of 2 hours are generally a sufficient indication of the mortality to be expected. It is interesting to us to see how infrequently ½ hour capillary blood sugar levels exceeding 200 mg. per 100 cc. of blood were found except when the 2 hour level was distinctly high. Nevertheless, it does not seem advisable for insurance purposes to omit either the initial or ½ hour samples because they generally yield valuable information.

With respect to the blood sugar limits permissible for standard insurance, our findings suggest that the 2 hour limit can safely be placed at 110 when the ½ hour peak does not exceed 200. A small rating may be considered for those who are moderately overweight, and some attention should be paid to the weight history. Our experience with cases having a ½ hour blood sugar over 200 and a 2 hour under 111 is too small to indicate a definitive rating, but we believe that most of those cases with a high peak and rapid descent to normal levels are safe risks for standard insurance. If the peak is above 240 another glucose tolerance test may be desirable.

When the initial or fasting level is between 121 and 140, but the rest of the record and current examination satisfactory, no rating is required, especially if less than 4 hours have elapsed between the last meal and the beginning of the test.

One must be sure, however, that there are no indications that the applicant is diabetic and that a dose of insulin has not upset the results.

For cases having 2 hour blood sugar between 111 and 120, with other levels satisfactory, our experience has not been within standard limits, and possibly some rating is required. Beyond this level and up to 160, and if the initial blood sugar is satisfactory and the ½ hour level is not over 200, only substandard insurance is warranted for those of average weight. These rules might be extended to those having a 2 hour blood sugar between 111 and 120 with ½ hour levels between 200 and 240. Overweight cases require larger ratings, and perhaps are not insurable. Attention is again called to the importance of the history of change of weight. All other cases either should be considered diabetic, particularly if they are overweight or there is an adverse weight history, and rated as such according to company policy, or should be requested to have another test.

As for insuring diabetics, we do not believe that our study gives a direct answer, even though the mortality ratios on cases having blood sugar readings considered as diabetic by some experts have been within the range of the substandard limits used by many companies. We say this because there is real doubt in our minds as to how many of these cases are truly diabetic. Of course, it must be remembered that these are cases presumably without symptoms and certainly without obvious hypertension, cardiac disease, or other serious impairments, and they are all in standard occupations.

A vital consideration in this matter is the recorded mortality from diabetes in our experience. We are well aware of the limitations of mortality statistics in this regard, but the studies made by Joslin in Massachusetts (13, 14) and by Palmer in Washington (15) show much higher proportions than ours of known diabetics recorded as dying from or with the disease. Thus, in Joslin's experience, based upon patients

of the George F. Baker Clinic, and dealing with four separate periods from 1926 to 1943, this varied from 72 to 80 per cent. In Palmer's experience based upon patients of the Mason Clinic during 1928–1944, it was 84 per cent. In contrast, less than one-fourth of the deaths in our high blood sugar group were due to diabetes, either primary or secondary, and only 10 per cent of the deaths in the borderline group. The highest proportion of deaths with or from diabetes in our experience was less than two-fifths, this occurring in the group having 2 hour blood sugar values exceeding 200. How may this difference be explained? Our cases expected to pass an examination for insurance, whereas those in the clinical studies were patients. It is probable that we are dealing in part with a group having a high percentage of early and yet undiagnosed diabetes. Many of our cases with high blood sugar at 2 hours may have had slow absorption of sugar or retarded utilization of it.

The fact that we have observed a higher than normal mortality from the cardiovascular diseases among our cases with elevated blood sugar suggests a possible relationship, but this may properly be left for the consideration of the clinician. Apart from this, we must not forget that about one-third of our applicants refuse to take the test when offered, doubtless because they know they have diabetes. This self selection is reflected not only in the distribution of our cases according to blood sugar groupings but also in the mortality results.

We confess that we are somewhat disturbed, perhaps unduly, by the high frequency of serious complications in diabetes of long duration, as reported by Dolger (16) and, for juveniles, by Joslin's group (17).

At any rate, juvenile diabetics cannot qualify for insurance unless there is further substantial improvement in their mortality. For, as compared with death rates of less than 1 to 2 per 1,000 prevailing in our standard experience at durations as high as 10 years for issues under age 30, the death rate among Joslin's patients (18) is over 5 per 1,000 at age 15; over 7 at age 20; and about 10 at ages 25 to 35.

We are, therefore, content to judge cases with high blood sugar on their merits and not make the diagnosis of diabetes in the absence of complete evidence or an authentic clinical history. We do not insure frank diabetics. We shall all await with interest the results of the analysis of the experience of companies which do insure them. In response to our recent questionnaire, it was learned that 46 out of 117 companies are now accepting them for insurance, as compared with 71 that still refuse them.

SUMMARY

The glucose tolerance test using capillary blood samples has proved itself as a selective method in the insurance medical examination.

We need not fear that a request for the test will discourage too many applicants. In fact, most of those who refuse it may be diabetic. The urine alone does not supply us with convincing information regarding the carbohydrate metabolism. The results of analysis of the Metropolitan experience on applicants given a tolerance test is reported in detail and the factors influencing the results are discussed. Diabetes was not the leading cause of death in any of the groups into which the material was divided. On the basis of this study, tentative proposals are made for the rating of applicants according to the blood sugar findings.

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PRESIDENT JIMENIS—Gentlemen, our next speaker needs no introduction to you. He has enjoyed half a century of outstanding work in the field of diabetes, has received many honors in recognition of his work, and is now Director of the George F. Baker Clinic in Boston, and Clinical Professor Emeritus of the Harvard University Medical School. Dr. Joslin!

DR. ELLIOTT P. JOSLIN—Dr. Jimenis' paper is a challenge to every medical director and every doctor who is concerned with the diabetic. It is true our two groups look at the problem from a different point of view. The medical director wishes to know how long a patient will live, but the doctor must decide whether the patient has diabetes and what treatment he needs.

Dr. Jimenis' data are based on a follow-up of 98 per cent of 5,532 cases. This shows the thoroughness with which he has attacked the problem.

We are glad to see that the interval since the last meal before the glucose tolerance test was extended from 2½ to 4 hours; our rule is 5 hours. We use capillary blood sugar values, particularly with children, but we have far more confidence in the venous blood sugar. We realize that excellent data can be achieved with a blood sugar fasting, ½ hour, and 2 hours, but in our work we prefer an additional blood sugar at the end of one hour because it adds confirmation to the others and we are ever so anxious to avoid placing upon an individual a diagnosis of diabetes unless that is really justified.

Like Dr. Jimenis, we consider a capillary blood sugar of 200 mg. about 30 mg. more than a venous blood sugar of 170 mg. What the relation would be between a capillary blood sugar of 110 mg. and a venous blood sugar of 110 mg. at the end of 2 hours we have no idea. The figure of 200 mg. capillary blood or 170 mg. venous blood is the dividing line which both Dr. Jimenis and we employ. He considers it practically the highest limit of normal, but we consider it as the lowest limit for a diagnosis of diabetes. We believe in holding to

this, because in our last analysis of patients we found so few errors when we followed that rule. However, Dr. Jimenis' recent research makes it incumbent upon us to look over every single record where a diagnosis was based on 170 mg. venous blood or 200 mg. capillary blood to note whether the future confirmed or disproved its reliability.

A reason why we use a venous blood sugar of 170 mg. as a diagnostic value is that so often we have random blood sugars at 2, 3 and 4 hours after food, but accompanied with glycosuria. I am sure Dr. Jimenis in such cases would agree in diagnosing diabetes. A difference in time element of 60-90 minutes is of supreme importance, and too often there may be time errors in the minutes consumed by the patient in drinking the 50 grams of glucose mixture. This always should be made up to 240 cc. or else it will be too concentrated.

Therefore, we feel that a capillary blood sugar of 200 mg., venous 170 mg., is a generous gesture toward the applicant or the patient. Contrariwise, our feeling would be that insistance upon a 2 hour blood sugar of 110 mg. would be ultra conservative. Such a value certainly by venous blood would be a trifle less. We believe the safety accompanying a 200 mg. capillary blood sugar at ½ hour rests on the 2-hour 110 mg. capillary value.

Heartily we agree with Dr. Jimenis that a little glycosuria is harmless unless backed up by a notable increase in blood sugar. Yet I must put myself on record that we never fail to follow up such cases.

With Dr. Jimenis we also agree that a blood sugar test is essential for a diagnosis of diabetes, but we believe the paucity of tests made in his wonderful series proves the need for expanding the number of tests and, incidentally, utilizing a cheaper and quicker method. When I picked out a doctor from the audience of onlookers at our exhibit at the recent A. M. A. convention, had his blood and urine examined for sugar, and found him a diabetic in five minutes, it taught me

a new era of diabetic diagnosis had arrived. Yet, he was a prominent surgeon, wholly ignorant of having the disease, living more than a thousand miles away.

Today all our non-diabetic patients have a blood sugar test at the first visit and often at subsequent visits. We cannot afford to miss a diagnosis, and I assure you when we discover an unsuspected case after years of non-diabetic treatment, it is a solace to have an array of previous normal blood sugar values.

The fact is that neither Dr. Jimenis nor we claim there is any arbitrary dividing line, and the only way to proceed with such patients is to make repeated examinations of the blood and urine in the next few weeks, months and years. And it is a fact that if this is done, one will see transitions between diabetic and non-diabetic states.

On Monday of this week I saw a patient, first examined eleven years ago, who then had a history of balanitis, 0.4 per cent sugar, a peak value of 170 mg., and a two-hour value of 100 mg., whose glucose tolerance test ten months later was normal and as yet shows no diabetes, although the venous blood sugar one hour after a meal was 168 mg. However, the very next patient, first seen four years ago, with glycosuria and a blood sugar reaching 209 mg. and a two-hour value of 168 mg., has never since had abnormal chemical or physical signs or symptons of the disease. A third patient immediately following the other two was similar to the second. I mention these three cases because they occurred on one day this week. One I thought diabetic, but he would have been considered by Dr. Jimenis a safe risk, and rightly so to date, but the other two would have been instantly declined by him and were considered frank diabetics by us. And yet by maintaining controlled body weight they have not developed evidence of the disease.

The above three cases illustrate the fallibility of the diagnosis of diabetes by glucose tolerance tests. Nevertheless, they do not invalidate the conclusions of Dr. Jimenis regard-

ing the life expectancy of a diabetic with his standard value of 200 mg. or less at the end of ½ hour and 110 mg. at the end of 2 hours.

In Dr. Jimenis' Table 4, in his normal class, may there not be a certain number of diabetics included, and may this not explain why the mortality in the subsequent follow-up was a trifle increased?

The 374 fat cases bring up a question. Is it fair to draw the same inference from 50 grams of glucose given to a person weighing 200 pounds as to a person weighing 100 pounds? I wonder whether some of the fat people who passed the test successfully were not far nearer the stage of later diabetes than those in the thinner group.

Causes of Death. These are hard to interpret. One wonders if, besides the 19 who died of diabetes among the 246 fatalities, there are not many others who had diabetes but did not succumb to it. The figures are based on small numbers, but from the fact that in the borderline group there was expected only one death from diabetes as against four reported, and between one and two expected in the high group as against 11 observed, there is an implication that this was the case. This is also favored by the fact that no deaths from the disease were recorded among the applicants under age 40 at issue. Young diabetics live long. It is certainly striking that cancer was so prevalent—one-sixth of the cases of both the substandard and declined groups. Contrariwise, the absence of any deaths from tuberculosis is notable, because so frequently such a cause exists in a diabetic clientele. The high percentage of cardiovascular deaths in the group declined fits in with our results. Surely it would be advantageous if these 246 deaths were followed up to determine whether the patient died with diabetes even if not of it. That would contribute immeasurably to the whole question. The lower mortality in the Metropolitan group is added proof that the million unknown diabetic series, if only diagnosed and treated, will be a still more valuable clientele for insurance.

Diabetes and Insurance. May I be allowed the privilege of making a few comments upon the diabetes situation in 1947 and its relation to insurance? We are entering into a new diabetic world and one in which life insurance companies will play a far more prominent part. Their issuance of insurance to diabetics already is exerting an influence upon our patients. It stimulates them to take better care of their diabetes so as to secure a favorable report from us for you medical directors. Thus you gentlemen are helping to place upon the patient a responsibility for his own care.

The Oxford Diabetic Survey demonstrated that for the million known diabetics in the country there were a million unknown. And this unknown, unrecognized million diabetics in general are the mild, symptomless diabetics who are especially amenable to medical care and destined, if diagnosed and treated, to narrow the existing difference of 25 per cent of life expectancy between diabetics and the community as a whole. Life insurance companies simply cannot disregard a possible 1,000,000 new risks. Moreover, this sizeable group of risks will be discovered for you by the Government and Boards of Health, but chiefly I trust by doctors, and the million known diabetics themselves who will find them among their relatives, particularly among their fat relatives. Insurance companies can aid in the program of discovering new cases without arousing criticism.

Prevention of the disease, diabetes, however, I believe to be the chief altruistic function of the insurance companies. You gentlemen can saturate the public with two ideas—first, that diabetes is hereditary and, second, most common in the fat relatives of diabetics from early middle life on. You can push still further your offer to do tests of urine and blood annually for all your policyholders.

Arteriosclerosis. Arteriosclerosis is uppermost in your minds and ours as a cause of death of diabetics. It is true that 67 per cent of our 651 deaths since January 1, 1944 have been of this character. Tragically enough, among young diabetics

of long duration its presence is high. Among 200 children with diabetes of twenty years or more duration and just analyzed by Dr. Priscilla White, we know it can be found in 85 per cent in their eyes, in 75 per cent in their blood vessels, and that 50 per cent will show renal lesions, 8 per cent coronary trouble and some 3 per cent signs of cerebral arteriosclerosis. But please note these figures are the worst possible figures which ever will be published, because they are based on a period when insulin was either not available or was given in insufficient quantity and when the diet was notoriously inadequate. Therefore, we believe that the pessimism which is creeping into the literature about arteriosclerosis in diabetes should be offset. We know that we have 16 diabetics who began diabetes in childhood and now, so far as arteriosclerosis is concerned, are perfect in their eyes, as certified by expert ophthalmologists, the absence of arteriosclerosis in their vessels, as proved by roentgenologists, and that they have sound kidneys. This patient who volunteered to allow me to show him to you as an example of this hopeful group, began his diabetes at the age of 14 in 1920 and meets all the requirements of 100 per cent freedom from arteriosclerosis. As proof of his physical stamina, I would point out that a year and a half ago in a railroad accident he had ten broken ribs, a puncture of the lung, shoulder blade broken in four places, saw the radius protruding at his wrist and yet his surgeon said he convalesced as rapidly as though he were a non-diabetic.

In the five viewing cabinets I want to call your attention to examples of calcifications in the course of diabetes which hitherto have seldom been described. First, a calcified vas deferens. Dr. Marks, the roentgenologist at the New England Deaconess Hospital, now has 17 such instances and only one occurring in a non-diabetic. Second, calcified pelvic arteries. The four cases shown are our children now 21, 27, 28 and 30 years of age who have had diabetes respectively 11, 13, 20 and 20 years. Calcified pelvic arteries are of enormous prognostic value in pregnant diabetic women, as

shown by the studies of Dr. Priscilla White. Thus, if the pelvic arteries are calcified, the chances of securing a living baby are reduced to one in four or even one in five. These pelvic vessels show calcification so extreme that in one of the films you can see the calcified vessel through the skull of the fetus in the woman's pelvis. Dr. Marks tells me that although one gets calcified pelvic arteries in our young diabetic males as well as young females, he has had but one example of calcified pelvic vessels in 75 comparable non-diabetics.

In the last 49 years I have lived through many pessimistic eras in diabetes. Once all the children died of diabetic coma, but now diabetic coma among our diabetic deaths in or out of the hospital is down to 3.1 per cent. Of the actual cases of diabetic coma treated in the hospital, only between one and two per cent succumb to it. The morbidity from carbuncles was high. Nearly every other patient died and now among our last 651 deaths there was not one death from a carbuncle and, in fact, thanks to modern sulfa drugs and penicillin, an operation upon a carbuncle is almost unknown. Gangrene has dropped from 8.1 to 2.9 per cent. Bouchardat never saw a pregnant diabetic woman in 1875, and yet Dr. Priscilla White has 32 now under supervision, and of the last 375 confined, there has been but one death and that was six weeks later from hepatitis. Even up to January 1, 1936 the death of the fetus occurred in about half the cases, but now between 90 and 97 per cent live, according to the hormonal condition of the mother and its treatment. But what encourages me most of all, both for the future of my diabetics and the future of diabetic treatment, is the steady improvement in expectancy of life as shown by the compilation of at least 24,000 of somewhat over 31,000 patients I have treated. In analyzing the age group, the 10 year old, 20 year old and the 30 year old, you will perceive that there has been no lapse of improvement in a single one of the six chronological periods beginning with 1897 and ending in 1945. Again I wish to express to the Metropolitan Life Insurance Company and the members of its Statistical Department the appreciation of our

group for compiling these records which represent our own follow-up of 99 per cent of the 24,000 cases on which the chart is based.

The policy of treatment of these 24,000 patients from the beginning to the end of this nearly one-half century, as shown in the chart, has been undeviating and can be summed up as an aggressive attack on the disease with an endeavor to control glycosuria and its antecedent hyperglycemia. Naunyn's dictum has been the guiding principal, namely, that the apparently severe case if vigorously treated often will surprise one later by becoming mild, but the mild case of diabetes neglected may eventually become severe. Confirmation of the soundness of this doctrine has recently been demonstrated by Dohan and Lukens. They proved that hyperglycemia was so harmful that if artificially produced in a cat for 2 weeks, degeneration of the islands of Langerhans would follow and actual permanent diabetes would result. The moral is clear: if hyperglycemia for two weeks will cause diabetes in an animal with a sound pancreas, how much more serious must its effect be if it is allowed to persist in a diabetic patient with a pancreas already damaged?

President Jimenis—You have heard this enlightening address of Dr. Joslin. Is there any other discussion of this subject? Are there any questions that you would like to ask?

Dr. Paul H. Langner, Jr.—I first want to congratulate you and your colleagues, Dr. Jimenis, on your splendid paper. I would like to say, too, that it has been very gratifying that these authors have confirmed the findings of Dr. Dewees and myself. Some of you may recall that in 1942 we presented the results of our studies on glycosuria and the glucose tolerance test, and at that time we concluded, on the basis of a group of cases we had followed, some of them for as long as twenty years, that renal glycosuria was entirely harmless provided one could be entirely sure that the blood sugar studies were normal.

I would like to say a word, too, on our procedure in the cases where the blood sugar tolerance is borderline or has been found by others to be so. We routinely use capillary blood in our studies, but in questionable cases, or where there is a great deal at stake, we prefer venous blood at fasting, ½ hour, and 2 hours, and also we give the applicant a little warning about what to expect and about antecedent diet which we believe is very important.

We have found that occasionally an applicant will think the right thing to do is to abstain from all carbohydrates for two or three days before the test, thinking thereby to improve his tolerance. Well, of course, just the opposite is true in a normal individual, and these people who come in a state of semistarvation have a consequent abnormal blood sugar tolerance test.

I just thought it would be of interest to make these two points. Thank you very much.

Dr. Harold W. Dingman—Dr. Joslin, which is the more favorable individual—he who has his diabetes so mild that it is controlled by diet, or he who is a moderately severe diabetic and takes insulin?

Dr. Karl W. Anderson—Dr. Joslin, I wonder if you would say a few words as to what constitutes a controlled diabetic. Our company is one of the companies that insures diabetics.

Dr. Joslin—I can answer that question, fortunately, on account of the work of Dr. White, and there is in the foyer, I think, a little chart which shows it. You cannot tell whether a diabetic is controlled or not when he comes in the hospital, because he either diets too much on purpose to make a good showing or he overdiets thinking that he is going to be treated vigorously, or, finally, if he has a complication it so appears worse.

What is the greatest common divisor in controlling it? No one likes diabetic coma. Dr. White has classified the children on the basis of whether they have had coma or not. She has taken those children who had no arteriosclerosis at all, or

just the barest hint of it in their eyes, and, if I remember the figure right, it is 17 per cent that have had coma at some time in the preceding twenty years.

Then she has taken the children that have had moderate arteriosclerosis, quite a good deal of it perfectly obvious with x-rays and by other means as well. They have had coma in about 40 to 50 per cent of cases, whereas of those who had incapacitating sclerosis and are blind, or were confined to bed with coronary artery disease, over 70 per cent have had coma. So, there is a measure of the control of the diabetic.

There are three of the controls which she has worked on. It holds essentially the same whether the blood sugar has been over 400 mg., that is, a marked hyperglycemia, where the blood cholesterol has been high and where the liver is large. So, there are actually four standards for measuring the control of the diabetic.

Mr. H. H. Marks—In closing, it seems advisable again to call attention to certain aspects of our study which, to a degree, reflect differences in points of view of clinical medicine and life insurance medicine. Our main concern, as in all underwriting problems, is to ascertain the deviations from the normal mortality in groups that are as homogeneous as it is practicable to constitute them, except in regard to the characteristic under observation. It is true that in this study we are dealing with quantitative observations but as we know only too well, not only does the blood sugar level of normals vary from individual to individual but in normal individuals there may be considerable degree of fluctuation from one reading to another. Consequently, in classifying our material, it was not our intention to display generosity or conservatism. We were simply applying the test of the mortality table to the various divisions of our material to find out what deviations from normal mortality existed. Our results were in no way an attempt to set limits of normal, although they may throw some light on this question. Thus, we are in no

position to say whether a ½ hour capillary blood sugar level of 200 mg. per cent is the limit of normal or not. We do know that in all the groups up to that point, we experienced an approximately normal death rate among our cases. We do not doubt that some diabetics may be included in this group but they were not sufficiently numerous nor severe that they raised the mortality ratio to an appreciable degree. In the same way, our division of the data with respect to 2 hour capillary readings at 110 mg. was not made out of conservatism but simply to find out what the mortality was around the level which generally was accepted as the upper limit of normal. We were as surprised as anyone to find that the mortality in this borderline group was high. We cannot be sure whether or not this was a chance variation. Only additional observations by us or by other companies will give the answer, but one must entertain the possibility that the upper limit of normal blood sugar at 2 hours has been set too high. In this connection, it may be noted that in a follow-up study* made several years ago by us, in association with Dr. Joslin's group, of cases originally diagnosed as non-diabetic glycosuria, we ascertained the proportion of those subsequently developing diabetes in various groups classified according to the blood sugar level at original observation. We found that as compared with those with fairly normal levels, there was a sharp increase in the proportion developing diabetes for the borderline group, namely, those with 2 hour blood sugars of 0.11—0.12 per cent.

With regard to the causes of death, we do not doubt that diabetes is under-reported in our study. However, because our information comes largely from official sources or is checked with them, and moreover, is often supplemented from other sources, it is unlikely that the degree of understatement would be any greater than appears in official mortality statistics generally. We believe, therefore, that little would be gained by further inquiry on the subject and that

* Marble, A., E. P. Joslin, L. I. Dublin and H. H. Marks, "Studies in Diabetes Mellitus–VII Non-Diabetic Glycosuria," American Jour. Med. Sci. Vol. 197, p. 533, 1939.

our findings with regard to causes of death have real significance.

There is no doubt that we would be on safer ground if we had more than one test but that is generally impracticable in the life insurance business. It is comforting to know, however, that in those cases on which we have had occasion to make more than one test, the results of the first test show a high correlation with those of subsequent tests, the exception being in the borderline cases. Where possible, it is probably desirable that an additional test be given in these cases.

We are all extremely grateful for the help that Dr. Joslin and his associates have given us in elucidating our problems in the underwriting of applicants with glycosuria.

PRESIDENT JIMENIS—Our next subject, arterial hypertension, is to be presented by Dr. Irvine H. Page, well known for his work in this field. He was formerly with the Rockefeller Institute and the Eli Lilly Laboratories and is now Director of Research at the Cleveland Clinic. Dr. Page!

THE NATURE AND TREATMENT OF HYPERTENSION

IRVINE H. PAGE, M. D.

From the Research Division of the Cleveland Clinic Foundation, Cleveland, Ohio

Your invitation to discuss arterial hypertension with you offers me an opportunity I could not but accept. Few groups of physicians are so importantly placed as you in making decisions which will affect the future of reseach in cardiovascular-renal disease. Your companies lose financially far more from its ravages than from other diseases. You are often the first to discover the disease and almost certainly the last to pay for it. Therefore, I need hardly point out that cardiovascular-renal diseases are the Number One Killer. I should not have to point out that from the viewpoint of the investigator, few diseases receive so little monetary support.

As a group who knows that broad, productive research costs money and a great deal of it, I would neglect my duty not to stress to you that the situation, despite some help, is very far from healthy. Support from the U. S. Public Health, Service has been helpful, but is on an annual basis. It is difficult and often unfair to younger men to offer them research jobs on such tenuous security. Your own Life Insurance Medical Research Fund seems to be headed in the right direction, though handicapped for lack of sufficient monies and for proper integration with those actually working in the field. So serious has the situation become that the American Foundation for High Blood Pressure was formed by a group of business men with the sole purpose of subsidizing long term research in hypertension and arteriosclerosis. It is hoped that this will be business' answer to the problem. Further, it is to be hoped that the American Heart Association will

also take its place in aiding these great purposes and that the time will not be far off when all of these agencies will fuse or co-operate to achieve the common goal.

It is not too much to believe on the basis of present knowledge that the outlook is bright for significant advance. Inviting vistas open on all sides beckoning for investigation.

But I have been asked to talk on the pathogenesis of hypertension rather than its economic and social aspects. Instead of attempting a penetrating analysis of certain special aspects of the problem, I shall try to give a unifying point of view, no matter how personal this may be. For without proper selection and orientation of particulars, we shall be unable to arrive at any universals so necessary to the Aristotelian dictum of what constitutes science.

First let us describe the problem requiring solution. Examination of the eyegrounds shows the arterioles to be constricted and the retina to be normally perfused: examination of the heart shows it to be beating with greater than normal force. Further, it will be noted that the skin of the patient is warm and normally perfused with blood. In this triad lies the nub of the problem of hypertension. The peripheral arterioles are constricted, the heart is beating more forcefully to drive blood at a higher pressure through them and it is all done in such a way that most peripheral tissues receive their normal complement of blood. Many more defining criteria may be added, such as, for example, the fact that cardiac output is not increased, but they are not usually necessary for an understanding of the problem.

In summary, then, we would define essential hypertension as a vascular disease of unknown etiology with diastolic hypertension in which blood flows in normal volume against increased arteriolar resistance associated with increased cardiac effort.

Pathogenesis

It must be evident that there are a variety of diseases in which arterial hypertension occurs and a classification of these is helpful in the analysis of patients with essential and malignant hypertension in which the cause or causes are not surely known. It has proved useful to view pathogenesis as a problem in which multiple factors enter, a mosaic in which an interplay of these factors yields the final clinical picture. In one patient the nervous factor may predominate, in another the renal, and so on, but always modified by other systems of the body. This variable interplay accounts in our opinion for the protean manifestations and outlook of patients with arterial hypertension.

The Nervous System

Evidence of neurogenic participation comes largely from the bedside. The irritable, hostile, or contrariwise, sometimes the docile patient with labile autonomic nervous system, can hardly fail to impress the attending physician with the importance of the nervous system. Other signs or symptoms of nervous origin such as blushing, sweating, headaches, lacrimation, tachycardia, transient paralysis, paresthesias, etc., are common. It is probably due to the lack of objectively measurable changes that has led to minimizing its participation in the genesis of the disease.

From the experimental viewpoint, perhaps the most impressive simple demonstration of neurogenic participation is destruction of vasomotor nerves by destroying the spinal cord in an animal with experimental hypertension. Immediately the blood pressure falls and stays at low levels for long periods. The converse side of the picture is exhibited when the hydrostatic pressure within the skull is artificially raised as in the Cushing experiment. Here the arterial pressure rises until perfusion through the cerebral vessels is reestablished. But despite the clear demonstration of nervous participation in the control of blood pressure, so far it has

Table I

POSSIBLE SYSTEM INVOLVEMENT IN ESSENTIAL HYPERTENSION

Neurologic Panel

Headache
Blushing
Sweating
Irritability
Lachrymation
Tachycardia
Cardiac Pain
Transient Paralysis
Confusion
Paresthesia
Stroke

Endocrinologic Panel

Hair and fat distribution
Menstrual disturbance
Diabetes
Abnormal urinary steroids
Increased excretion of steroids
Edema
Salt retention

Vascular Panel

Compensated and decompensated vascular sclerosis of eyegrounds, heart and kidneys

Humoral Panel

Renal efferent arteriolar constriction
Increased cardiac effort
Vasoconstriction in eyegrounds
Necrotizing arteriolitis

not been possible to produce persistent neurogenic hypertension in animals which mimics essential hypertension. Two varieties of hypertension have however been produced. The first, by section of the carotid sinus and aortic depressor nerves, leads to chronic hypertension, but the hemodynamic changes are quite different from those of essential hypertension. It differs chiefly in that cardiac output and rate are markedly elevated with minimal increase in peripheral resistance, which is the reverse of that in essential hypertensives. The second, by inducing cerebral ischemia with constricting ligatures of the great cerebral blood vessels. While very moderate hypotension occurs, it is not impressive nor does it last more than several months. The degree of ischemia required to produce any elevation of blood pressure at all is very great.

The participation of the psyche in the regulation of blood pressure needs no special pleading. Nor need it be stressed that all those factors which quiet the mind, in a word, the eliciting of equanimity, reduce arterial pressure and improve the well being of the patient. The problem remains how to achieve it in the patient and further how to produce experimental hypertension of psychic origin in animals.

Attempts are now under way in a variety of institutions to employ different types of psychoanalysis, guidance, hypnosis, etc., to achieve the end of peace of mind. The therapeutic results have varied widely so far. It is much too early to form an opinion as to the value of such procedures.

The chief source of information on the participation of the nervous system in the genesis of hypertension has come from the operation of sympathetic neurectomy and ganglionectomy on hypertensive patients. A hesitant and uncertain beginning had been made in the use of these operations on the continent as long as 20 years ago, but it was not until about 1933, when they were actively investigated as a method for the treatment of hypertension, that any significant observations appeared. We need hardly remind an audience of physicians of the wide

variety of opinion expressed on the results of these operations, having run the gamut from "a cure" to "positively harmful". But time and observation have softened the differences. Five facts of importance may now be regarded as established (1) arterial pressure in some patients is significantly lowered (2) the patient is clinically improved when fall in blood pressure occurs (3) symptomatic relief may be obtained without significant fall in arterial pressure (4) no seriously harmful effects have been observed as a result of the fall in blood pressure (5) the operation is not a minor one and in a small number serious complications or even death has occurred as a result of the operation. These results suggest the important participation of the nervous system in the genesis of hypertension. They do not, however, prove that it is primary.

The Endocrine System

Ample clinical evidence proves the participation of the endocrine system in both the maintenance of normal arterial pressure and the genesis of special types of clinical arterial hypertension. For example, removal of the pituitary or adrenal glands in normal animals results in chronic hypotension. Injection of desoxycorticosterone acetate while rats are being fed high salt diets, or in rats with glomerulonephritis, result in nephrosclerosis and hypertension. Clinically somewhat similar counterparts are found in the hypertension associated with Cushing's syndrome, adrenal cortical carcinoma, and adrenal pheochromocytoma. The hypertension accompanying Grave's disease is of a very different nature, being due chiefly to the increased cardiac output incident to hypermetabolism of the cardiac muscle. Insofar as we can determine, belief in menopausal hypertension is ill founded. Rather, hypertension appears commonly at the age when menopause also occurs. There seems to be no casual association.

The signs and symptoms of endocrine participation are subtle but in some few patients, outspoken. Hair and fat

distribution may be abnormal, menstrual disturbances, diabetes, abnormal quality and quantity of urinary steroids, salt retention and edema may indicate abnormal endocrine function. Objective methods of measuring endocrine participation in patients with essential hypertension are few. Still, some as yet not very exhaustive studies, strongly suggest the importance particularly of the adrenal glands, in the genesis and maintenance of elevated blood pressure. Suffice it to say that investigation in this phase has only made a beginning, but hopes for the future seem to us especially bright.

Renal—Humoral System

One hardly need be reminded that the kidneys have come into and out of favor as primary cause for essential hypertension since the time of Bright. It is generally admitted that the hypertension associated with pyelonephritis, glomerulo-

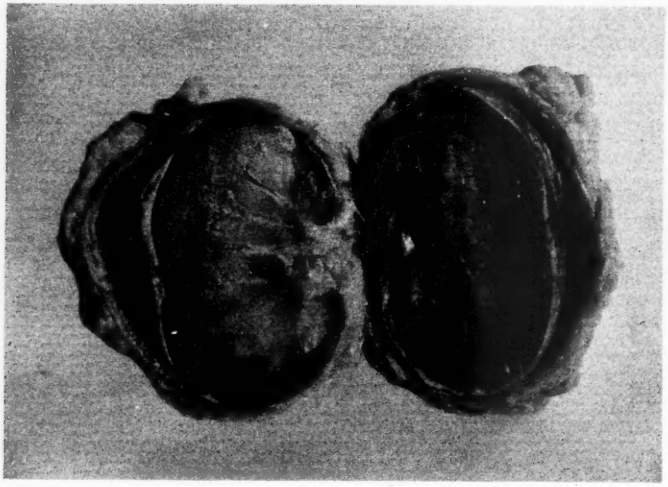

Figure 1

The perinephric hull which forms around dog's kidneys after being wrapped in silk or cellophane. The hull gently squeezes the renal parenchyma and elicits arterial hypertension without renal ischemia.

nephritis, and perinephritis almost surely is of renal origin. Experimental hypertension in animals, whether produced by ligature of large renal vessels, clamps on the vessels or perinephric constricting hulls resulting from the application of cellophane or silk to the renal parenchyma,. must be primarily renal in origin. But whether essential hypertension is so produced remains unproved and largely in the realm of authoritative opinion.

The kidneys contain a proteolytic enzyme most probably in the tubular cells which when secreted into the blood stream acts on a protein substrate contained in the α_2 globulin fraction of plasma to produce a powerful pressor substance called "angiotonin" in parts of this country and "hypertensin" in South America. The mechanism of the rise in blood pressure after its injection into animals or man is so similar

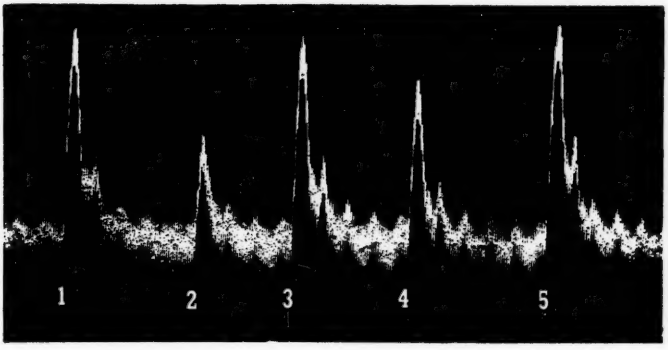

Figure 2

The quantitation of angiotonin in the pithed cat. Within reasonable limits the angiotonin response is directly proportional to the magnitude of the dose.

to that in essential hypertension there is good reason to suppose it is concerned in the mechanism of the elevated pressure in that disease. But the evidence is as yet incomplete, for

angiotonin has not yet been found in the blood of human hypertensives. Some evidence suggests that it may be present in protein-bound form in which the angiotonin molecule may be acting as a prosthetic group.

Since angiotonin is almost unique in the way in which it elevates blood pressure, and since similar mechanisms may be operative in the essential hypertensive, the significance of angiotonin seems evident.

Measurement of renal participation in the mechanism maintaining elevated arterial pressure is most unsatisfactory. It should be remembered that there is no demonstrable relationship between renal excretory efficiency and the height of the blood pressure. Thus tests of renal function based on ability to excrete, while highly useful in following the progress of damage to blood vessels, does not yield a measure of the activity of the renal pressor system.

At the present time, about the best that can be done is to measure the intrarenal hemodynamic changes by means of p-amino hippurate for tubular and inulin or mannitol for glomerular function. The increase in cardiac effort can be arrived at only indirectly. Many investigators have attempted to evaluate separately the contribution to the maintenance of arteriolar constriction of the nervous system and of the humoral system. Spinal and caudal differential anesthesia and tetraethyl ammonium chloride are currently under study for this purpose.

Treatment

1. *General Measures.* In a short space, it is not possible to describe in detail the general measures so important in the treatment of hypertensive patients. This has already been done in a recent manual for the patients themselves.

Perhaps the principles of these general measures can be summed up as follows: (1) Cultivating serenity; (2) coming to terms with the inevitable; (3) living a life of moderation; (4) participating only in those affairs which one can influ-

ence; (5) avoiding fatigue; (6) having more frequent periods of rest; (7) avoiding obesity; (8) avoiding food fads and eating a well-balanced diet in small repasts unless specifically advised by the physician to do otherwise; and (9) selecting a physician in whom the patient can place full responsibility for wise counsel. Each of these measures requires much thought and planning, and when carried out thoroughly and systematically, will add much to the comfort of life and probably conserve life itself. Administered in a cursory fashion, both patient and physician lose invaluable aids. Properly administered a significant fall in pressure may be anticipated.

Excessive nervousness contributes greatly towards keeping the blood pressure elevated. Its control is often a complex problem. If, as often happens, it is associated with the female menopause, administration of 0.3 to 0.5 mg. of Stilbestrol daily with meals may do much to relieve it. Occasionally, it is due to marked hyperthyroidism, when it should be treated as any other case of this disease. Phenobarbital (0.03 Gm., ½ grain t.i.d.) is a generally useful sedative and may be continued for a long time if necessary. The barbiturates are very satisfactory when used under the physician's guidance for insuring adequate sleep. Other more elaborate physical methods such as "progressive relaxation" will not be discussed in this short review. Psychoanalysis or psychiatric guidance has its place in the treatment of some patients.

If hypertension occurs in association with one of those rare diseases such as tumor of the adrenal gland, clearly the treatment consists in removing the exciting cause. Less than five per cent of all patients will come under this category.

2. *Nephrectomy.* If it is demonstrated that disease is limited to only one kidney, its removal has been observed in some cases to be followed by return of the blood pressure to near normal levels. The basis for nephrectomy is unfortunately not so simple as this would lead one to believe. There is no known method which demonstrates that one kidney is ab-

solutely normal and the other diseased. The impression is too common that reduced excretory function in one kidney and normal renal excretory function in its partner constitute convincing evidence. Actually, there is no direct relationship between excretory efficiency and height of the blood pressure as we long ago showed. Nor does there appear to be any clearly defined relation between hypertension and the appearance of the kidney as demonstrated by the pyelogram. Secondly, the conclusion has been drawn from very brief experiments in rats and dogs that the removal of the kidney causing hypertension is always followed by a return of blood pressure to normal. When the latter has been elevated for months or years, removal of the offending kidney is ordinarily not followed by a return to normal. Much the same seems true in human beings. So there has been in the past few years a false optimism that nephrectomy would be a very important tool in the treatment of hypertensives. Perhaps this optimism is now giving way to a wave of too great pessimism.

Thus, if it is shown by x-ray examination or by kidney function tests that one kidney is obviously infected and that hypertension has developed in the past two or three years, and that the other kidney seems normal, it is probably desirable to remove the offending kidney. The principal indication for nephrectomy in such cases should be urological, rather than as a treatment for hypertension. If the hypertension has persisted so long that it is accompanied by manifest arteriosclerosis, or if the evidence for unilateral disease is uncertain, it would appear better to avoid the operation, unless there is a clear urological indication.

As an example of the remarkable effects nephrectomy sometimes produces in early cases of hypertension, that of MacKay, Proctor and Roome may be given. This patient underwent a pelviolithotomy and while still in the hospital, the arterial pressure began to rise. Shortly, this was followed by the signs of severe malignant hypertension. Because of the alarming course of the disease, nephrectomy was decided

upon. At operation it was found that a thick hull enveloped the parenchyma from which the kidney could be shelled out. Shortly after nephrectomy, the blood pressure returned to normal and all of the signs disappeared.

In general, it is wise to view nephrectomy as a procedure which should be done when for reasons other than the hypertension, removal of the kidney would be desirable. Only occasionally, a patient is seen in whom abolition of the hypertension is the prime object of the operation.

3. *Potassium Thiocyanate:* This salt has had a checkered career in the treatment of hypertension. It was introduced many years ago but fell into disrepute because of toxic manifestations occasionally observed. When M. H. Barker published a method for controlling the dosage by its level in the blood, a new wave of interest occurred. Since that time, the drug has been extensively studied. Despite this, there are two schools of thought about its value.

Many are convinced that it has a real place in the treatment of hypertensives. It lowers arterial blood pressure moderately in roughly 30 to 35 per cent of the patients if optimal levels of thiocyanate are reached in the blood stream. This is after the decline in pressure usually resulting from more general management. It often is a most valuable remedy for intractable headaches that afflict hypertensives. This appears to be its especial virtue.

Its drawbacks consist chiefly in the fact that it often causes a feeling of intense lassitude, of heaviness of limb. Eruptions of the skin, and more especially, the mucous membranes, may occur. In older patients, mental disturbances have occasionally been encountered. Death from thiocyanate has even been recorded in the literature, but analysis of such records seems to indicate unwise judgment in the use of the drug in some of these cases. It is fair to say that death has occurred at some time from almost every drug in common use.

Apparently it is now a minority who believe thiocyanate is valueless in the treatment of hypertensives. They contend that the mechanism of its action is unknown. This is true. Its action is said to be a purely "toxic" one. This argument plays loosely with language because the term, "toxic", is not sufficiently precisely defined. It is granted that thiocyanate lowers pressure moderately in 30 to 40 per cent of cases and subjective improvement occurs; but the latter may occur in patients receiving thiocyanate in whom no lowering in blood pressure occurs. This improvement is therefore believed to be "psychic". There is also no constant dosage at which either toxic or therapeutic effects may be anticipated. This is only partially true. All agree that the drug is irregular in its action, but it is usual to obtain therapeutic response at blood levels of from 8 to 12 mg. per cent thiocyanate in the blood if one is to be obtained at all. Above 16 mg. toxic manifestations commonly occur.

Thiocyanate may be administered in solution or as tablets. Enteric coated tablets often dissolve irregularly in the gut, leading to varying blood levels. Differences in the observed response of patients may well be due to the care with which they are observed and perhaps more importantly to the control of the level of thiocyanate in the blood. The level had best be measured by a reasonably accurate colorimetric method.

Our own experience with some 500 patients treated with the drug is that it is moderately useful. We have seen only occasional patients who showed signs of its severe toxic action and none of them have been serious. No deaths have occurred. It has been not uncommon to see patients who were said to respond unfavorably to thiocyanate and who show no toxic signs and even a favorable response when the use of the drug was carefully controlled.

4. *Sympathectomy:* Dorsolumbar and splanchnic nerve resection as methods of treating patients with hypertension, just as thiocyanate, have been greeted with cheers or jeers.

It is probably true that reasons initially offered for the performance of these operations were incorrect. And it is very doubtful to me that any better ones have subsequently been offered. As a result, a flood of criticism greeted the early work. Time has shown much of this to be unjustified. The field was further confused by those who attempted to transfer results obtained on animals with experimental hypertension directly without demonstration of the validity of the transfer to human beings, concluding that since in dogs these operations produced little or no reduction in arterial pressure, the same was true in man. The pioneering experiments were harshly criticized, but now there is a tendency to be uncritical and overly optimistic of the results of operation.

Probably, fortunately, sympathectomy for the treatment of hypertension developed empirically and before hypotheses were advanced to explain it. In fact, if it had depended on some of them it might never have been practiced. Thus, it has been suggested that the effect of the operation does not depend on denervation of the vasomotor apparatus of the abdomen other than the kidneys, but rather on the relief of renal ischemia. In this view, renal denervation alone should be fully effective, whereas it has no effect. Further, the view depends on renal ischemia as the efficient cause of hypertension, whereas such ischemia is not uniformly present either in experimental hypertension or in hypertension in human beings. Lastly, the operation only rarely increases renal blood flow, which is usually unchanged after an otherwise satisfactory operation. The fact that renal blood flow does not usually decrease after sympathectomy when arterial pressure has fallen, indicates that renal resistance must have diminished, and, although without reference to the hypothesis of renal ischemia, it has also been suggested that this fact establishes a beneficial and specific effect of renal denervation. This point of view too is defective in that it ignores the normal autonomy of the renal circulation by which the kidney varies its resistance with arterial pressure in order to maintain as well as can be a normal rate of blood flow.

There is no reason to suppose that this mechanism is in any way defective in hypertensives and, indeed, we have good evidence to establish its presence and nearly normal activity. Persistence of this intrinsic renal mechanism of regulation of blood flow after operation can scarcely be attributed to denervation. Sympathectomy only leaves the kidney where it was before and its effectiveness in lowering blood pressure is therefore largely extrarenal.

We have briefly discussed sympathectomy from the viewpoint of what has added to knowledge of the mechanism of the disease. Now we would add a word as to the usefulness of the operation. There is now no doubt that when these operations are sufficiently extensive, as in the technic of Smithwick, the modified Adson procedure, or the even more complete Grimson operation, that marked fall in both systolic and diastolic pressure occurs in many patients. This is most pronounced when the patient stands erect. Indeed, postural hypotension is one of the best indices of the completeness of the sympathectomy. The length of time blood pressure remains reduced is variable. The average is perhaps from three to five years, some less and others more.

It is not unusual for regression of the morbid changes in the eyegrounds of patients with malignant hypertension to occur. And one of the most striking changes is the loss of headaches and regaining a sense of well being.

One of the greatest difficulties in the application of the method has been the inability to find any single or even multiple tests which determine whether a favorable outcome is to be expected. The hypotensive effect of administration of sodium amytal has been most extensively used to ascertain the drop in pressure to be anticipated as the result of operation. Some believe that when an adequate fall in pressure does not occur, the likelihood of success is poor, but that an adequate fall is no guarantee of success. Various authors have their own criteria and it now seems to be a matter of personal experience as to how patients are selected.

As we said before, the precise mechanism of the action of these operations is not clear. It is not improbable that several factors play a part, among these being a reduction of venous return to the heart as a result of denervation of extensive vascular areas. Besides this, the denervation of the large splanchnic area prevents the normal reactive vasoconstriction from occurring when the patient moves from a horizontal to an erect posture. Fainting is often observed, presumably because this protective mechanism has been blocked in its action.

Thus it may well be that the overall reduction of blood pressure during any 24 hour period may be quite significant and the time taken away from the destructive and sclerosing effects of the elevated pressure on the blood vessels contributed towards increasing longevity.

5. *Kidney Extracts:* The reasons for the search for substances in the kidneys which might lower blood pressure need not concern us here. Extracts of kidneys have been prepared which lower blood pressure and cause improvement in the clinical condition of patients, especially of those with malignant hypertension. But the mechanism by which these extracts act is entirely unknown.

The term, "nonspecific", has been employed to describe their action. This may be true in the superficial sense of the word, i.e., the lowering of pressure is due to an unknown mechanism set into action by a heterogeneous group of substances. Among these is fever, but many patients have fever without reduction in arterial pressure, and vice versa. The important point to recognize is that if any form of therapy will lower blood pressure and benefit the patient, it does not make a great deal of immediate difference what the mechanism is.

It is the belief of a very few investigators that certain types of extracts of kidneys have beneficial effects, especially in the management of malignant hypertensives, but none to date has been able to prepare an altogether suitable extract. Such

a search is naturally a tedious and expensive job, since in the last analysis, patients must be the test objects and nothing is known of the chemical nature of the substance sought.

There is evidence which suggests but does not prove some degree of specificity. Kidney extract will reverse the intrarenal hemodynamic change usual in most cases of hypertension to a more normal one. Further, cardiac output will be elevated in hypertensive patients when the mean pressure falls.

It is quite clear that work along this line is still in its embryonic stage. None can foresee its outcome, hence the desirability of not attempting to codify knowedge in this field prematurely.

6. *Vitamin A:* As the result of the clinical report of Villaverde and Pena, large amounts of vitamin A have been administered to hypertensive patients in this country. But our results have shown clearly that vitamin A of itself, even in great amounts, does not lower arterial pressure. In hypertensive animals, some samples of fish body or liver oils seem to lower arterial pressure. The investigation of this phase of the problem has not progressed sufficiently far to justify an opinion as to its value.

7. *Excessively Low Sodium Diets:* Twenty years ago Frederick Allen, and even long before him, Ambard, recommended diets containing from 200 to 500 mg. of sodium chloride for the treatment of hypertension. For several years thereafter controversy raged, with the verdict finally against the salt-free diet. The controversy was not without its bitter moments.

Two years ago, Grollman again revived the subject, suggesting reduction of sodium intake to 0.5 Gm. or less. In young plethoric patients, the reduction in blood pressure was marked as compared with the slight reduction in the older arteriosclerotic hypertensive. The clinical experience presented was very meager.

More recently, Bryant and Blecha claim that a diet of 2200 calories containing 70 Gm. of protein and 200 mg. of sodium gives results better than sympathectomy and similar to those of the rice diet. This paper will receive wide and possibly uncritical attention because of the hope it offers to those who wish to avoid operation. It should be pointed out that the evidence presented carries little conviction. Their best results were with the older group of hypertensives in contrast to Grollman's results.

Clearly, a period of enthusiastic advocation of a wide variety of dietary treatment is being initiated. More than usual caution must be observed in the gathering of evidence to support the claims of benefit. Further, let us hope that we shall remain like physicians of old belonging either to the schools of Cos or Cnidos which differed little on the principles of medicine, rather than to the Dogmatics and Empirics, so dividing medicine's practitioners into obstinate and unruly factions.

8. *Rice Diet*: This diet of 400 Gm. rice, fruit juice and sugar (2000 calories, 20 Gm. protein, 460 Gm. carbohydrate, 5 Gm. fat and 200 mg. sodium) has been tried extensively throughout this country in the past few years, but few reports on the results have appeared. No final judgment can therefore be made at this time, but apparently there is a growing belief that the diet is of little value in the early stages of hypertension except insofar as it removes excess weight. In early phases of malignant hypertension or the uremia of Bright's disease, the result may at times be dramatic. The diet is unpalatable and boring; consequently, it is difficult to persuade patients to remain on it for the prescribed period. Its too prolonged use may lead to some reduction of renal blood flow due to lack of sufficient protein.

At the present time, it is particularly difficult to evaluate the diet fairly because of great emotional bias generally being demonstrated by both patient and physician. Whether rice protein has any special virtue remains to be proved. How

much of the supposed benefit is due to the low protein content of the diet, the low salt content, both, or the authority with which it is fed, also remains for unbiased study. It is to be hoped that Kempner himself will provide the answers and that other investigators will not be deflected from their own attempts to understand and treat hypertension. The scientific capital of the field is too meager for its uneconomic deployment.

Let it be repeated that substituting large numbers of inadequately studied patients for fewer numbers carefully studied only adds further confusion. The error of omission consists chiefly in insufficient study before an experimental treatment is started. Despite the fact that many investigators have stressed the lability of the blood pressure, the warning continues to go unheeded. It is common experience to observe a fall of 50 mm. Hg. or more during the first week of observation in the hospital, and even thereafter, marked spontaneous changes may occur. Until the trend is established, best by twice daily blood pressure measurements, and the degree of lability ascertained, it is dangerous to draw conclusions about the effect of experimental treatments. Even more uncertain are results drawn from insufficiently repeated outpatient examinations.

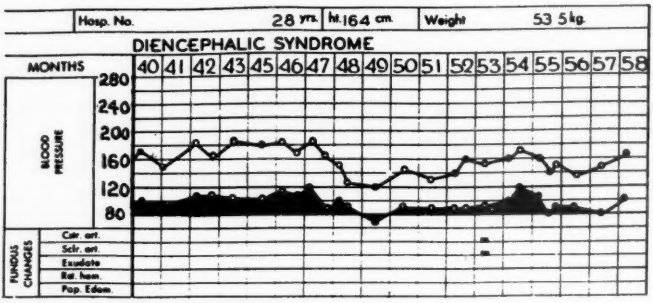

Figure 3

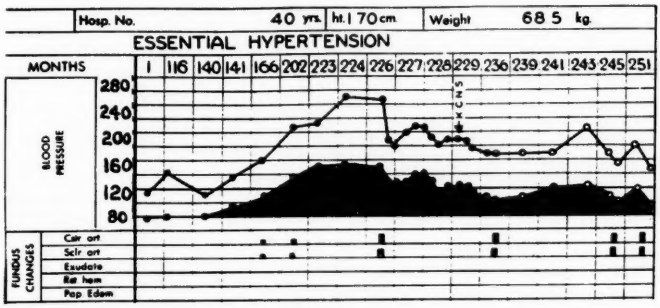

Figure 4

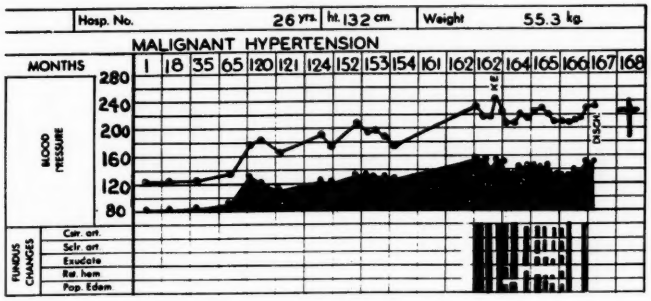

Figure 5

Figures 3, 4, 5

Charts of patients illustrating the wide variability of arterial blood pressure even when average measurements are dealt with rather than single measurements. Each dot represents the average of 14 determinations, the pressure being measured twice a day for one week, except in the chart labelled "diencephalic syndrome" where the open circles are single measurements. It is important to note that the prognosis is very different in these three patients which could hardly have been predicted from the blood pressure chart. Further, it is evident that unless great care were exercised, an experimental therapeutic regime administered during one of the spontaneous changes might easily have badly misled the investigator.

9. *Rutin:* This substance prepared from flue-cured tobacco or buckwheat is believed to reduce capillary fragility in man. Unfortunately, it has been touted in the lay press as a treatment for hypertension itself. There seems to be no evidence whatever for this claim.

The problem of measurement of capillary fragility is proving to be a serious stumbling block in the evaluation of rutin. Many investigators believe the Gothlin index and tests similar in principle to be unreliable. Thus it is difficult to assess work based on these methods. Clinical observation suggests that in some forms of purpura, rutin slows or stops the skin manifestations.

It is purported to be of value in preventing cerebral hemorrhage. At present, there seems to be no cogent experimental evidence to support the claim. It is doubtful, to say the least, that most cerebral hemorrhages begin or are concerned with fragility of the cerebral capillaries; therefore, even if their fragility could be reduced, it is unproved that apoplexy would be avoided.

It is now also claimed that rutin decreases capillary permeability of the blood-aqueous barrier and so is of value in the management of glaucoma. There is only one thing certain and that is that the enthusiasm of commercial advertisers has so far outrun the facts, that millions of dollars worth of rutin will follow the millions of dollars worth of vitamins down the gullible gullets of the American public.

10. *Pituitary Irradiation:* According to Pendergrass, Griffith, Padis and Barden, about one-half of 93 patients with essential hypertension showed significant reduction in arterial pressure months to years after irradiation of the pituitary gland. All were selected because of positive tests for serum antidiuretic hormone. They believed that an even higher per cent of success could be achieved, if besides the positive serum antidiuretic test, a negative test for serum gonadotrophic hormone and normal renal function were observed. Further, that a second dose of x-ray be given three months after the first in case the test for antidiuretic hormone did not become negative.

It is too early to arrive at any fair evaluation of these results but there are certain suggestions that might aid, both at present and in the future. The assumption that a pressor sub-

stance—presumably pitressin—is the cause of the elevated blood pressure in essential hypertension has received no cogent support since it was suggested years ago. Further, Pendergrass has shown that the presence of antidiuretic hormone and hypertension do not parallel one another. It would appear more likely that any effect irradiation might have would be by lessening the secretion of adrenocorticotrophic hormone. It is known from experimental work that this is the mechanism by which blood pressure is lowered in renal hypertensive dogs and rats after hypophysectomy. Aside from mechanism, perhaps the chief objection to acceptance of the work is that no adequate data concerning the fluctuations of blood pressure before irradiation are given. It deserves repeating again and again that particularly in the early stages of hypertension, wide changes occur in an almost unpredictable manner. Without presentation of these data, it is impossible to form an objective view as to the success or failure of treatment. This applies equally to much of the work on sympathectomy and probably more than any single factor has retarded unbiased evaluation of the value of operation.

Treatment of Heart Failure in Hypertensives

While it has long been recognized that decreased cardiac output in relation to the needs of the body for blood is one of the important mechanisms of congestive heart failure, work in the past few years has rather stressed the participation of sodium retention in controlling the size of the extracellular fluid space. The hydropigenous action of sodium has been known for many years and it has been custom in treating patients with Bright's disease and edema to restrict salt and allow unlimited amounts of water in the hope that thus more sodium would be washed out of the body and hence less water retained in the extracellular fluid space as edema. Schemm found somewhat the same regime useful in the treatment of congestive failure. Cardiologists, insufficiently un-

aware of the mechanism of the important participation of the sodium ion had restricted its intake inadequately and further had restricted water intake as well.

It is now usual practice to restrict salt to amounts of about 1 Gm. per day and allow fluids from 3 to 5 liters a day. The diet must be adjusted to the taste of the patient and not simply to that of the dietitian. Desalted dried milk preparations such as Lonalac, are convenient and palatable. There is also no substitute for chemical determination of the sodium chloride excretion in 24 hour urine specimens as a control on the patient's intake of salt. Possibly, the majority of patients are unable, without repeated periods of guidance, to maintain a low salt diet. It is not improbable that the majority of salt-poor diets do not work simply because they are not salt-poor. An uncontrolled diet is worse than none at all.

There can be little doubt that the use of a low salt diet in congestive failure has a sound theoretical background. The most recent contribution to it, that of Burch, Reaser and Cromrich, has pointed out that salt permeates the vascular wall freely and in the average man 50 pounds per day cross this wall back and forth chiefly into the interstitial fluid. In patients with congestive failure, the diffusion from the vascular bed is even more rapid and the clearance of it by the kidneys is much less than normal. It is easy to see that with only very slight disturbance in diffusion and in clearance, how sodium might be retained in the extracellular fluid space so leading to edema.

Some patients with congestive failure, particularly of long standing, with "cardiac cirrhosis", have lowered plasma proteins. This leads to lowered osmotic retention of water in the blood stream and its easier egress into the tissues. Sometimes the hypoproteinemia is correctable by raising the protein content of the diet. Unfortunately usually it is not, if only for the reason that few sick cardiacs are able to eat diets containing much protein.

Further, there seems to be a growing concern with the inadequacy of the Starling hypothesis which has been so vigorously exploited in this country in the past years. Landis' results of intracapillary pressure measurements which appeared to offer strong support, are according to Chambers and Zweifach, inapt since no account was taken of the opening and closing of the capillary valves, or the normal topography of the capillaries. Thus, in contrast with Landis, Chambers places less stress on protein osmotic pressure and more on the nature and activity of the capillary bed. It will remain for the future to determine which view is the more nearly correct. For our purposes, the example is used to show that the mechanisms of edema are far from adequately understood.

Conclusion

Views on the nature and treatment of diseases of the circulation, arterial hypertension in particular, are rapidly changing. The pessimism of twenty years ago is giving way to the hope that with increased understanding of the mechanism of these diseases, cure may ultimately be achieved. Cure is rarely achieved today, but much can be done to ameliorate the disease.

The pathogenesis of hypertensives remains unsolved, but investigation is actively being pursued on the three principal theories, namely (1) the participation of circulating pressor agents (2) increased vasomotor activity and (3) endocrine participation. The fusion of these seemingly diverse beliefs may well occur.

Treatment must cover a wide variety of signs or symptoms because blood vessels are affected in most parts of the body. No specific remedies which will lower blood pressure are available but the discerning use of several agents leads to far greater comfort for the patient and doubtless in many cases to prolongation of life.

PRESIDENT JIMENIS—Dr. Page, we have enjoyed your talk tremendously, and I think we have learned a great deal from it. Is there any discussion of this subject, and are there any questions that you would like to have answered?

If there is no discussion, we will announce a recess until after lunch. We will meet here again at two-fifteen.

Our session this afternoon opens with a paper by Dr. Bradley L. Coley, the title being "Prognosis in Tumors of Bone and Sarcomas of Soft Tissue." If time permits, Dr. Coley is also going to speak to us about "Trauma in Malignant Tumors of Bone." Dr. Coley is Attending Surgeon at the Memorial Hospital, Bone Tumor Department, and also Director of Medical Education at the Memorial Hospital. In the late war, he served as colonel in the Medical Corps and saw service overseas. His new book on "Neoplasms of Bone and Related Conditions" will be off the press early in 1948. Dr. Coley!

PROGNOSIS IN TUMORS OF BONE AND SARCOMAS OF SOFT TISSUE

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Among those whose duty it is to pass upon the suitability of applicants for life insurance, the question must not infrequently arise as to the risk involved in accepting one whose application lists among previous operations or illnesses a neoplasm which had its origin in bone or soft tissues. The latter group includes muscle, fascia, fat, and nerve elements, in addition to sites in or about joints as well as bursae (synovial membrane). Or if the application fails to define the true nature of the neoplasm, then the question of insurability will arise after the company has received information from the attending surgeon or from the hospital concerned.

It is my purpose to touch upon those benign and malignant tumors that are most frequently encountered and to explain briefly their prognosis. In order to simplify matters for my audience, I shall list them in tabular form as follows:

Tumors of Bone

Benign	*Malignant*
Osteoma	Osteogenic Sarcoma
Osteochondroma	Chondro-sarcoma
Central chondroma	Ewing's sarcoma
Benign chondroblastoma	Multiple Myeloma
Angioma	Reticulum cell Sarcoma
Fibroma (non-osteogenic)	Malignant Giant cell tumor
Solitary Bone Cyst	Metastatic Cancer of bone.
Giant cell Tumor (benign)	
Adamantinoma	
Chordoma	

Tumors of Soft Parts

Lipoma
Fibroma
Angioma
Neuronevus
Benign pigmented nevus (mole)
Xanthoma of Tendon Sheath
Neurofibroma
Benign tumor of Synovial origin
Leiomyoma (uterine fibroid)
Fascial Myxosarcoma
Neurogenic Sarcoma
Myosarcoma
Liposarcoma
Synovial Sarcoma (Synovioma)

Benign Tumors of Bone—Let us consider first the benign tumors of bone. Osteoma, angioma, fibroma and bone cyst are consistently benign and when removed the patient is not liable directly or indirectly to any future hazards on their account.

Osteochondroma and central chondroma are originally benign but in a definite although relatively small percentage of cases a malignant transformation (into chondrosarcoma) may later take place. This does not happen after a complete extirpation of the tumor while it is still in the benign stage but we have seen it occur following an incomplete removal. We believe that the same may be said for benign chondroblastoma of bone.

Angioma of bone is rare. We have not seen it undergo malignant degeneration. Giant cell tumor is more unpredictable. Certainly the majority of cases that have been treated successfully by surgery or by moderate doses of roentgen-rays, are completely cured and future difficulties are not to be anticipated. However, in perhaps from 8 to 10 per cent of cases a gradual change in the character of the tumor takes place, and the recurrent tumors reveal a more cellular structure

which may attain truly malignant histologic features. Such transition requires us to classify giant cell tumors in this situation as malignant neoplasms fully capable of metastasis and a fatal outcome. I should therefore take the position that a person who had had a giant cell tumor, histologically benign, that had been removed surgically five or more years previously, who was symptom-free and whose films at the time of application for insurance showed a stationary process, could be regarded as a good risk. On the other hand if there had been several operations with recurrences then such a person would have to be regarded as liable to future serious difficulties.

Adamantinoma of the jaw, while usually considered a benign lesion, is difficult to control save by radical jaw resection at the outset. It tends to recur and to grow slowly. It may have been present for upwards of 20 years. It rarely metastasizes. Such tumors render the subjects doubtful risks over the long stretch.

Chordoma can be placed in a similar category. This tumor, owing to its location either in the sacrum or at the base of the skull, does not lend itself to complete extirpation. Therefore most of the patients succumb to the direct or indirect effects of it, and it too on rare occasions may give rise to metastases.

Malignant Tumors of Bone—Osteogenic sarcoma has too serious a prognosis to make one willing to accept for insurance a patient who has had an amputation for this condition, unless there has been about a ten-year interval of freedom from recurrence. "Five year cures" are glibly reported but we have seen death occur later from this disease in about 8 per cent of our five-year survivals. Chondrosarcoma follows the same pattern and can be considered with the osteogenic group. In a consecutive series of 89 cases of osteogenic sarcoma and chondrosarcoma treated by amputation we have had about 30 per cent five-year survivals.

Ewing's sarcoma (endothelioma) has a frightful prognosis.

Probably less than five per cent of the patients with this disease are actually cured. Over a period of twenty-five years we have recorded less than half a dozen successful results.

Reticulum cell sarcoma of bone presents a better outlook. This is a rather rare tumor. We have in our files 24 cases of which 11 were treated prior to 1942; of these 6 are alive and apparently free of any signs of the disease from 5 to 21 years.

Plasma cell myeloma (multiple myeloma) is a disease which is at present completely incurable. The same is almost as true of metastatic bone cancer.

Benign Tumors of Soft Parts—It is difficult to express an opinion as to the significance from the standpoint of future possibilities of some of the benign soft-part tumors.

Small lipomas generally carry no serious threat even if not removed; if completely excised there is usually no further difficulty. Large lipomas may undergo a malignant change, appearing as myxoliposarcoma, a lethal neoplasm.

Fibromas (usually neurofibromas) may become sarcomatous; they are then know as neurogenic sarcomas, which extend, metastasize and cause death.

Patients with Recklinghausen's multiple neurofibromatosis are known to be prone to the development of neurogenic sarcoma in one or more of the tumors.

Xanthomas (giant cell tumors) of tendon sheath origin do not carry any serious threat, and patients with these tumors should not be refused insurance. The same excellent prognosis holds for women who have had a hysterectomy for fibromyoma of the uterus. Angiomatous soft-part tumors may be considered as likely to remain benign.

Malignant Soft-Part Tumors—Both fascial sarcomas and neurogenic sarcomas, while frequently causing death from metastasis, are curable in a fair percentage of cases by radical surgery, i.e., wide extirpation or amputation. Five years' freedom

from evidence of disease following such treatment renders the patient's future outlook extremely promising. Myosarcoma, often a more serious condition, is likewise curable by surgery in some instances. Here, too, when the five-year period has elapsed the prognosis may be considered favorable.

This is not the case with synovial sarcoma (malignant synovioma), for here, late fatalities are not at all unusual, even after a period of ten or fifteen years. I should never recommend accepting for insurance a person whose condition has been diagnosed as malignant synovioma by a competent pathologist.

Liposarcoma is a tumor which carries about the same risk as myosarcoma; it usually metastasizes within three or four years after treatment.

It should be borne in mind that there are certain bone conditions which are of themselves benign but upon which sarcoma may develop. Among these I should mention Paget's disease of bone (osteitis deformans), polyostotic fibrous dysplasia, and in particular, benign bone tumors, e.g., bone cyst and giant cell tumor which have been heavily irradiated. This latter group, the so-called *sarcomas on irradiated bone*, develop only after a considerable lapse of time, usually after 12 to 15 years. I should therefore hesitate about issuing insurance to a person who had been heavily irradiated with x-rays for any type of bone disease. While we must admit that there are less than 25 such cases reported in the literature, nevertheless our records include seven examples, and we believe that the incidence of this complication of roentgen therapy is more frequent than the published reports would lead us to suspect.

The subject of tumors and their importance in life insurance underwriting and claims settlements would not be complete without some reference to the related medicolegal problems. Recently, I published a paper[1] on the subject, a

(1) Coley, Bradley L.: Trauma in Malignant Tumors of Bone, Am. J. Surg. 73: 300–304 (Feb.) 1947.

portion of which I should like to combine with this discussion of the prognosis in bone and soft-part tumors.

**Trauma*—It is my feeling that in many medicolegal cases in the past, injury as a determining factor has been over-emphasized and that decisions have been rendered on a flimsy structure of scientific evidence. It is believed that the system in vogue at present whereby medical expert testimony is given, is shocking in several particulars and is rendered less valuable by the undeniable fact that the expert would not have been called by his side had his views not been known in advance and his opinion anticipated as being favorable. Until the present set-up is revised and all experts are called, employed and recompensed by the Court rather than by the plaintiff or the defendant, real experts will shun medicolegal cases and the evils of the present day will continue unabated.

In the interest of fair play, to assure a complete clinical record, to avoid important blanks in the sequence of a given history, it should be the inviolate rule that whoever takes the initial history of a patient with a suspected sarcoma should set forth in the records certain pertinent facts. First of all he should ask the patient to what he attributes his tumor and if the latter describes an injury, he should then be asked whether the cause has been suggested to him by others. Proceeding with the history it should contain the following facts: (1) Date of injury, (2) nature and mechanism of injury, (3) site of injury, (4) subsequent manifestations of injury, e.g., ecchymosis, swelling, tenderness, disability, (5) period following injury in which these symptoms persisted, and (6) interval between subsidence of symptoms and signs of injury and first recognition of symptoms or signs of tumor.

In addition a complete clinical record should include roentgenograms of the part concerned taken as soon as possible after the injury as well as others taken subsequently.

* We are indebted to the *American Journal of Surgery* for permission to reprint this portion of Dr. Coley's paper.

Aggravation. The provisions of the Workmen's Compensation laws of many states presume that an injury is compensible which can be construed as having aggravated a pre-existing malignant tumor. Therefore, claims of aggravation are often made, and this introduces a much more controversial issue. From what is known of the dissemination of malignant cells it is probable that direct trauma to the primary lesion might be conducive to metastasis. However, the only experimental evidence on single trauma in tumors of known dissemination rate fails to support this viewpoint, and the fact remains that malignant tumors do metastasize with or without local trauma. It has seemed to me that in a given case one would have to show that the clinical course of the disease had been adversely affected by the trauma, i. e., that it ran a much more rapid course than the average untraumatized case. Even then one must realize that there is a great natural variation in the rapidity of spread and total duration of malignant tumors. It would appear therefore that if a sarcoma were accepted as having existed prior to injury the circumstances of its course thereafter would have to be exceptional before one could justifiably assume aggravation.

Decisions in these accident cases that are adverse to the workman or to the injured plaintiff frequently entail severe hardship to the individual or his family. In an effort to avoid any possible injustice to the injured party the doctor is apt to let his sympathies color the medical testimony which he offers. This motive seems also to influence the referee, the jury and sometimes even the judge. Yet as Stewart has pointed out, this is no excuse for making awards in the face of incompetent evidence or of using "supposedly scientific medicine to further his Notion of proper social behavior."

Stewart illustrates the faulty reasoning of the "supposed expert" as follows:

"The same surgeon who will do all sorts of orthopedic jobs involving chiseling into bone or insertion of such objects as ice tongs or pins or screws may testify that a blow which has

left no real signs has caused an osteogenic sarcoma, although he never thinks his surgery will do so nor has he ever warned a patient with the severest form of bone trauma—a fracture —to be on the lookout for a possible sarcoma." Such arguments bear much weight and are hard to refute.

There are certain bone processes and benign lesions in which the rôle of trauma seems more difficult to deny. Of these the most conspicuous is benign giant cell tumor of bone. Considerable authoritative opinion upholds the theory that this tumor is commonly caused by the effects of trauma. Many patients with giant cell tumor have been awarded compensation, and we believe rightly so. On the other hand, it should be pointed out that pathologists who have had considerable experience in bone neoplasms are unconvinced that even in giant cell tumor trauma is an important etiologic factor.

On rare occasions we find an osteogenic sarcoma associated with a previously incurred ossifying hematoma and in such instances it is difficult to dissociate the origin of the sarcoma from the injury which caused the ossifying hematoma, and yet the periosteal hemorrhages of scurvy are apparently never the precursor of sarcoma.

Certain cases of osteochondroma or chondromyxoma have been studied where injury has seemed to initiate a change in the type of tumor to that of chondrosarcoma and occasionally unsuccessful extirpation of such a benign tumor has been followed by recurrence and a transition to fully malignant chondrosarcoma.

Therefore we believe that injury may play an important part in such transition and as a preventive measure we advocate removal of these tumors when they occur in a location which renders them liable to external injury. It must be admitted however that on a number of occasions we have seen such malignant degeneration occur in cases of osteochondroma in which there was no history of injury.

To summarize briefly, it would seem that the rôle of single injury in the development of bone sarcoma has been overemphasized. A critical evaluation of each case and a decision based upon a scientific appraisal of all the known facts has not been the rule. Little support of the *traumatic* theory has been afforded by the pathologist; no laboratory experience seems to favor it. Considering the universality of injuries on a par with those alleged to be the cause of sarcoma of bone it is difficult to explain why so few of them are followed by the development of sarcoma.

So-called expert testimony on matters involving the alleged relationship of injury to malignant disease is not at present likely to appeal to the disinterested observer as worthy of credence; it is colored by the profit motive. Until qualified authorities are summoned by the court and not by the plaintiff or the insurance carrier to pass upon the merits of each case in a judicial and scientific manner, medical expert testimony will continue to be of doubtful value and will not enhance the doctor's reputation.

In conclusion I should like to emphasize the following points:

1. There are certain benign tumors of bone which may undergo transformation into malignant tumors and are fully capable of causing death.

2. There are certain malignant tumors both of bone and of soft parts that can fairly be regarded as cured after a lapse of sufficient time following treatment. Any period less than five years is not considered sufficient, and in the more highly malignant tumors, such as osteogenic sarcoma, a ten-year interval is more certain.

3. Synovioma has an utterly bad prognosis even though death may not occur for ten or fifteen years after the first evidence of disease.

4. Plasma cell myeloma and metastatic cancer of bone are practically always fatal regardless of the method of treatment employed.

5. The rôle of single injury in the development of bone sarcoma and other malignant tumors has been overemphasized.

6. Qualified authorities should be selected impartially by the courts, rather than by other interested parties, to testify on matters involving the alleged relationship of injury to malignant disease.

PRESIDENT JIMENIS—Is there anyone who would like to ask any questions on bone tumors? If not, we will proceed to our next paper. This next subject is to be introduced by a man familiar to you all, Dr. Harold Fellows, Associate Medical Director of the Metropolitan Life Insurance Company, who has the ability to translate the results of special examinations into information useful in underwriting. You will recall his invaluable work formerly as Chairman of the Electrocardiographic Committee. Dr. Fellows!

DR. HAYNES H. FELLOWS—The idea of developing this part of the program really came about last year at this meeting, when at the forum the subject of Pulmonary Tuberculosis came up for discussion, and everybody was rocked back and forth with conflicting evidence and conflicting opinions. It seemed to me I left here somewhat confused by trying to figure out whether we had any possible answer to a question which was perplexing you and us. And so the next step in this business which will come about this afternoon was to send out a questionnaire to 55 representatives of the companies, asking what they did in various types of applications involving a question of pulmonary tuberculosis.

When I got back the questionnaires, I found that the previous confusion, which I hoped was temporary, was permanent. The life insurance industry showed no consistency, but one company would have one action on a given case and another company, or many other companies, would have different actions, entirely opposite.

Well, in the meantime I had asked our actuaries if they would try to find out the results of what we had been doing

since 1930. We did not know whether we were on the right track or what was happening. All these problems were milling around at once—questionnaires and the actuarial studies—so I did not get the answer to our experience until along about May or June.

In the meantime, I had asked two of our men who I knew had to make up their minds on this problem, "What are you going to do with an applicant who shows some evidence of tuberculosis or who says he has had tuberculosis?" I asked them if they would join me in bringing to you from three independent sources a review of the manner in which we approach these problems, hoping that if we did that something useful, either by proof or by discussion, might result.

This report, as far as I am concerned, is an obligation. Dr. Jimenis mentioned this morning that if we can determine a trend long before we have a finished study, that trend has a value. Dr. Joslin spoke about the Public Health Service making surveys of diabetics. I feel it a conscientious duty to report to the public, the life insurance industry and the practicing physicians what we have done so far.

In this country, upwards of 25,000,000 people have had chest surveys in the last few years. A large part of that public has applied for life insurance and has had to submit a film of the chest to a life insurance company. The public has a definite interest in knowing that when they submit a piece of evidence the insurance industry knows what to do with it. The life insurance companies that accept these applications and consider the x-rays have a right to know the limitations and possibilities because they will issue, rate, or decline based on a special examination. The practicing physician—and here is the thing that I want to stress—should understand and have it impressed on him that even if the life insurance companies can take large groups of people and secure a satisfactory experience based on actuarial tables, that is no indication whatever that a private physician can take a single film and make anywhere near an accurate prognosis as to what is

going to happen to an individual patient. I do not think it can be done.

With that much of an introduction to the subject, I would like to introduce to you my good friend, Dr. David Reisner. Dr. Reisner is Supervisor of the Department of Tuberculosis in the Department of Health in New York City. He has, among other problems, those dealing with employment, follow-up, and disposition. Then, there is Dr. Kiessling, of the Prudential Insurance Company, who has to handle this same type of material and who has a contribution to make. We have at the Metropolitan a little proof which may be determined as a trend.

Now I would like to introduce Dr. David Reisner. Dr. Reisner!

THE ROENTGENOGRAM AS AN AID IN THE DISPOSITION OF CASES OF PULMONARY TUBERCULOSIS DETECTED IN GROUP SURVEYS

DAVID REISNER, M. D.

Medical Supervisor, Bureau of Tuberculosis
Department of Health, City of New York

The remarks which I am privileged to address to you are made chiefly from the standpoint of the clinician and public health worker who deals with the tuberculosis problem. As you know, case-finding measures for detection of pulmonary tuberculosis have in recent years become recognized as one of the most important means in the control of this disease. It is equally well known that the volume of routine chest x-ray examinations is being expanded on a greatly accelerated scale to include ever-increasing segments of the general population.

As a result of the expanding case-finding programs, tuberculous lesions are being detected in many thousands of supposedly healthy individuals. Because of this, there has developed an increasing need for certain practical criteria which would be helpful for the evaluation of the relative importance of the various types of lesions that are being discovered. Usually, there is little difficulty in arriving at a proper decision in cases in which on the basis of symptoms, clinical, x-ray and laboratory findings, there are indications of an active process. However, experience has shown that such cases represent only a relatively small proportion of the total number of persons in whom tuberculous lesions are found by means of routine chest x-ray examinations. In the majority of cases confirmatory clinical or bacteriological data are lacking and the x-ray findings represent the only available indication of the existing lesion. This is especially true of those cases in which the lesion is of minimal extent. The fact that

in the experience of the New York City Health Department, approximately 70 per cent of all cases of pulmonary tuberculosis detected on routine chest x-ray examination were classified as of minimal extent, may serve as an indication of their numerical importance.

The principal problem which confronts us in detecting such lesions is the question as to what extent they represent a potential risk of progression to advanced and clinically manifest disease. The important practical considerations that present themselves in the disposition of individuals showing such lesions are as follows: (1) The need for treatment; (2) The possible risk connected with normal employment; (3) The indications for intensive and long-term supervision, either by the private physician or by public health agencies.

Since, in our experience, in the majority of such cases the clinical and laboratory findings are of little assistance, we have come to rely to a large degree on the x-ray findings as an aid in determining the relative significance of a given lesion at the time of its initial detection. However, in order to derive the maximum benefit from the roentgenological demonstration of the pulmonary lesion, we have found it essential to consider, in addition to the mere extent of involvement, the qualitative factor or character of the lesion. For it should be remembered that a tuberculous lesion in the lung undergoes from its inception certain evolutionary changes, either of a favorable or unfavorable nature, which may result either in progression or retrogression of the pathological process. While retrogressive changes with healing often take place as a result of treatment, there is ample evidence to indicate that quite frequently this occurs spontaneously in individuals who have never experienced symptoms of active disease. Spontaneous healing is more likely to occur in those cases in which the lesion has not extended beyond the minimal stage.

It is thus to be expected that pulmonary lesions discovered as a result of routine x-ray examination, may represent dif-

ferent phases of their evolution. Some of them may be truly early lesions whose future behavior is extremely uncertain and which often progress to advanced clinical disease. In a much larger proportion of cases the lesion demonstrated on the x-ray film represents a late form which at the time of its first detection has already reached a healed stage. Others may be found in various intermediary phases of development and thus represent gradations between the two extreme types.

Several years ago we made a study of a group of 469 cases of minimal tuberculosis diagnosed in the clinics of the New York City Health Department [1]. In the majority of these cases the lesions were originally detected on routine x-ray examination and the average follow-up period amounted to five years. In analyzing the data on this material we paid particular attention to the character of the lesion as it appeared on the x-ray film. In classifying the types of lesions we followed a pathological terminology. Although the use of pathological terms in interpreting x-ray findings is not generally accepted, we feel that, from a practical standpoint, this classification is essentially not different from the one which uses a purely descriptive roentgenological terminology, provided that the interpretation of the x-ray findings follows a basically similar pattern.

We have found that three major types of lesions could be distinguished, namely: (1) the predominantly exudative form, usually appearing as a "soft", either flocculent or homogeneous density and which is likely to be of recent origin; (2) the productive-fibrotic or fibro-calcific types, which on x-ray appear as "hard" nodular or stringy densities and which are likely to represent lesions of old standing with manifestations of healing; (3) a mixed form, termed exudative-productive or exudative-fibrotic, which may be regarded as an intermediate type between the two preceding forms.

(1) Reisner, D. and Downes, J., Minimal Tuberculous Lesions of the Lung: Their Clinical Significance, Amer. Rev. Tuberc., Vol. 51, 393, (May) 1945.

The results of our observations concerning the behavior of these types of lesions may be summarized as follows: In the preponderant majority of individuals with lesions of the chiefly exudative or "soft" character there was some evidence of change indicative of activity, either progression or regression. The rate of progression amounted to 50 per cent in white persons and to 60 per cent in non-white persons. In the intermediate category in which the lesions represented a combination of the exudative and fibrotic type, the rate of progression amounted to 30 per cent, or about half of the preceding group. The predominantly "hard", fibrotic and fibro-calcific lesions were characterized by a stationary or inactive behavior in about 90 per cent of the cases and among these, lesions of the fibro-calcific type showed the highest rate of stability, progression being a rather exceptional occurrence. These data are shown in Chart 1.

In emphasizing the importance of the x-ray findings, it is essential to point out that this does not mean that one should rely entirely on the roentgenogram, to the exclusion of other significant data which may be helpful in the *clinical* evaluation of the case. It is obvious that for a thorough appraisal of the lesion, both from the standpoint of diagnosis as well as regards activity and presumable prognosis, it is necessary to utilize all the available clinical and laboratory data, including adequate bacteriological examinations. The age of the individual and the racial factor must be given due consideration, since experience indicates a greater liability to progressive disease in younger age groups and in non-white racial groups.

In order to obtain the most information from the x-ray demonstration of the lesion, two basic requirements must be met, especially when one attempts to differentiate between roentgenological types of lesions. From a technical standpoint, films must be of uniformly superior quality and for proper interpretation of the findings thorough experience in chest diseases, especially in tuberculosis, is of paramount

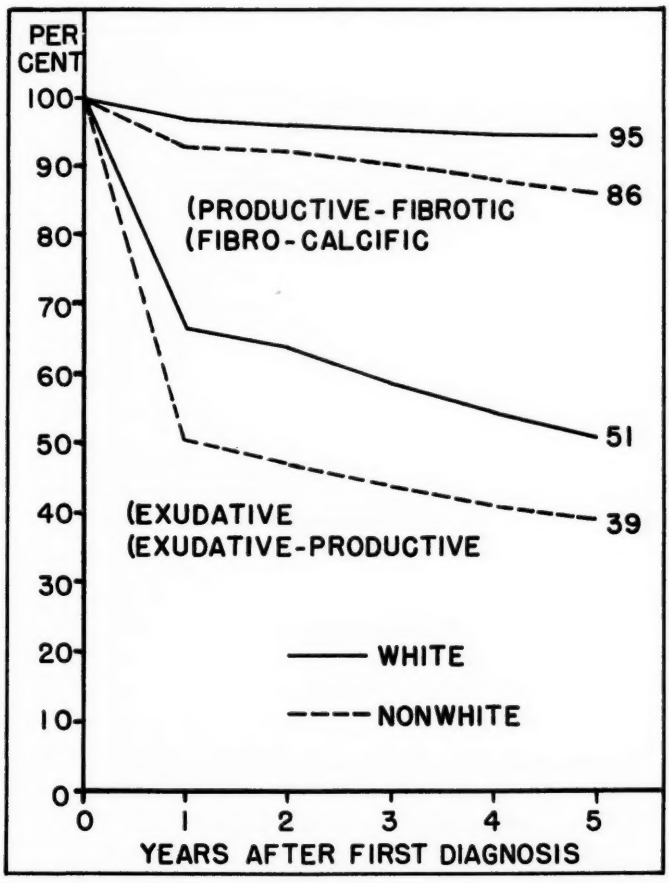

CHART I. PER CENT OF CASES OF MINIMAL TUBERCULOSIS SHOWING NO PROGRESSION OF DISEASE DURING FIVE YEARS AFTER INITIAL DIAGNOSIS OF TUBERCULOSIS.

importance. One should also keep in mind the fact that in many cases a single film is insufficient for accurate appraisal of the lesion and that additional views in other than the conventional position, as well as follow-up observation, are often necessary.

In conclusion, I wish to say that it is our belief that in spite of important limitations inherent in the x-ray method, the roentgenogram represents a most useful tool which, when properly and judiciously applied, is of inestimable value in the disposition of cases in whom tuberculous lesions are found on routine examination. Correct interpretation and evaluation of the findings may be used to great advantage in rendering proper advice to the individuals concerned and in preventing undue hardship to increasingly large numbers of persons.

SECRETARY KIRKLAND—In the temporary absence of Dr. Jimenis, may I call on Dr. Charles E. Kiessling, Assistant Medical Director of the Prudential Insurance Company. Dr. Kiessling!

SELECTION OF INDIVIDUALS WITH A PERSONAL HISTORY OF TUBERCULOSIS ON THE BASIS OF A SINGLE CHEST X-RAY

CHARLES E. KIESSLING, M. D.
Assistant Medical Director
The Prudential Insurance Company

Dr. Fellows will show you that on the basis of a single chest x-ray, and only on this basis, it is possible to separate individuals who have had pulmonary tuberculosis into insurable and uninsurable groups. Using our Home Office employees who have had pulmonary tuberculosis as subjects we put this idea to a test. Only those individuals who received treatment for active disease while employed at the Prudential were included.

Before telling you our results, I should like to describe this group in general terms.

It is only a small one of 82 individuals. However, its worth lies in the fact that we have rather complete records of each individual and most of them have been followed clinically by one of us.

Economically, they belonged to the low-middle and middle class income groups.

As you will see in the follow-up study, we have tabulated recurrences as well as deaths. Although we have observed these people for a period of 4–24 years with an average follow-up period of almost 10 years the ultimate mortality will naturally be worse than the mortality is now. How much worse I do not know, but I believe we are justified in assuming that it will roughly parallel the recurrence rate. Regardless, I feel that you will agree that any group with a high incidence of recur-

rences is an undesirable one from an insurance selection standpoint.

All of these individuals had reinfection type, parenchymal disease. The recurrences were diagnosed on the basis of a spreading lesion as shown on serial x-rays and in a large proportion of the cases by a positive sputum as well. They were actual recurrences and I do not believe we missed any.

Since 40 per cent of the Medical Directors who answered Dr. Fellows' questionnaire said they were giving consideration to applicants with a personal history of arrest for two years, that is, who could be classified as "apparently cured", we made our selection at the end of the two year period.

Among our Home Office employees there were 82 individuals with a definitely known history of having been treated for active disease, on whom we had adequate follow-up records, who were free from symptoms, who had been working steadily for two years, who had a lesion which appeared stable by serial x-ray examination, who were not receiving pneumothorax and who had not had a thoracoplasty. These were the criteria for admission to the study. They were theoretically potential candidates for insurance and they were considered as such.

They were not divided on the basis of the extent of their lesion but this was given consideration. The principal deciding factor was the character of the lesion as it appeared on an x-ray film. Individuals having a hard, sharply defined, calcified, or fibrous lesion were accepted and those having a soft, poorly defined lesion were rejected. In general those having an intermediate type of lesion were declined. It is very difficult, if not impossible, to put on paper or tell you where we drew the line between acceptable and not acceptable chest x-rays. The best I can do is say we acted conservatively.

An unidentified postero-anterior x-ray of the chest taken when the disease had been arrested for two years was placed

before me and, with no other information, a decision was made as to whether or not that person was insurable.

PERSONAL HISTORY
OF
PULMONARY TUBERCULOSIS
ARRESTED 2 YEARS (APPARENTLY CURED)

		RECURRED	DEAD
ACCEPTED	31	4	0
REJECTED	51	16	4

Of the 82 individuals in the series we accepted 31, roughly 40 per cent. Of those accepted, 4 had recurrences but none have died to date. Of the 51 we rejected, 16 had recurrences and 4 eventually died of tuberculosis.

To those of you who may be interested in the relationship of body build and recurrences, I shall say that 25 per cent of the accepted group were underweight and one of those who was accepted and had a recurrence was underweight. Of those we rejected 36 per cent were underweight and of those whom we rejected and had a recurrence 32 per cent were underweight. Of those who eventually died one was very much underweight, one was borderline, and the other two had normal weights at the time the x-ray on which the decision

was made was taken. These figures indicate that the factor of body build in this series was not decisive. Our selection disregarded body build entirely. It was made on the basis of the extent and character of the lesion.

We then repeated the experiment, using for the selection an x-ray taken 5 years after arrest. Only 70 individuals were included because 12 of the original group had recurrences before five years had elapsed and were therefore automatically excluded. A slightly larger proportion was accepted with 3 recurrences and a slightly smaller proportion was rejected with 9 recurrences, including 2 deaths. Time does an excellent job of selection in tuberculosis.

This is admittedly a very small series but it suggests what can be done; in fact it is being done every day by physicians who treat tuberculosis.

Given a group of individuals who have had active tuberculosis and who have reached that stage of recovery at which they can be considered "apparently cured", it is possible on the basis of a single chest x-ray to divide that group into two, one of which is insurable with a small rating, the other of which it appears wiser to reject.

Before I close I wish to thank my colleagues at the Prudential for their assistance and suggestions and to mention Dr. Lauritz Ylvisaker for his foresight in instituting the collection of this data which we have found so useful.

PRESIDENT JIMENIS—Thank you, Dr. Kiessling, for your splendid contribution. The final speaker on this subject is Dr. Haynes H. Fellows, Associate Medical Director of the Metropolitan Life Insurance Company. Dr. Fellows!

RELATIONSHIP OF X-RAY AND TUBERCULOSIS IN UNDERWRITING

HAYNES HAROLD FELLOWS, M. D.

Associate Medical Director

Metropolitan Life Insurance Company

This report is made with a sense of responsibility toward at least three interested parties.

A. The Public
B. The Life Insurance Companies
C. The Practicing Physician

The Public is interested because 25 million or so have been x-rayed in the last few years and many of them have been asked to submit their films when applying for life insurance. The Life Insurance Industry may issue, rate or decline an application depending upon the interpretation of an x-ray film and has a vital interest in knowing the possibilities and limitations involved. And the Practicing Physician must understand that even if the Life Insurance Companies can secure a satisfactory mortality experience on a basis of actuarial probability when dealing with *groups* of persons, there is no evidence that a doctor can make an accurate prognosis using a single x-ray film when dealing with an individual patient.

This is, as far as I know, the first report of the results of the use of the x-ray film as the most important and weightiest evidence in making an immediate disposition and a long range prognosis upon applicants for life insurance in whom there is a question of pulmonary tuberculosis. In this series of 1817 lives*, the x-ray films revealed either the presence of a known

* The accompanying chart and table show the mortality experienced by different groups of lives classified by x-ray. These lives applied for insurance in the Metropolitan in the years 1930–1941 and the experience terminated December 31, 1946. Applicants ineligible for Ordinary insurance by reason of occupation or impairment (other than tuberculosis or related impairments) were excluded from the study. War deaths from enemy action also were excluded.

and admitted lung lesion, disease which was not known or was not admitted, or an average healthy chest even though the applicant gave a history of having had pulmonary tuberculosis or an allied condition. Based upon the x-ray interpretation alone, the cases were placed in the following groups.

1. Far Advanced Tuberculosis
2. Active Tuberculosis
3. Healed Tuberculosis (probably more accurately "Inactive Tuberculosis")
4. Healed Primary Tuberculosis
5. Healed Pleurisy
6. Average Healthy or Normal Chest

The x-ray classification used to make this division includes two factors, one in use for years and the other not generally used or accepted except by physicians taking care of tuberculous patients. The *extent* of the lesion as defined in the Diagnostic Standards of the National Tuberculosis Association has been in general usage for years and is a measure of the amount of disease present. The other factor is the *character* of the lesion, and by character I mean the density, pattern, and structure of the shadows seen. It is not implied that the consideration of character is new or original because it is not. Physicians experienced in the care of tuberculous patients always take into account the appearance of the lesion and any changes present in guiding a "cure." We have incorporated both of these factors into a working guide to the immediate disposition and a long range prognosis. It is only fair to say that many doctors well versed in the interpretation of chest x-rays and with years of experience behind them do not believe it possible to predict future behavior with any accuracy on the basis of the interpretation of a single film. Our results to date indicate that it is possible to determine with relative accuracy whether the disease is probably healed (inactive), or safe; probably unhealed or active, and therefore unsafe; or of an indeterminate character and of doubtful activity. As a matter of fact, routine case-finding by x-ray

has made it obligatory to make some such preliminary and tentative decision even when we are handicapped by having a single film rather than a series of x-rays.

I would like to discuss briefly the *character* of the shadow. An active tuberculous lesion almost invariably has an appearance which arouses the suspicion of an experienced observer and as it heals the x-ray appearance changes. An early active lesion usually casts a "soft" or cloudy shadow which may be large or small, confluent or scattered, mottled or homogeneous. As it becomes inactive and heals, the lesion is more sharply outlined and is opaque or "hard" if calcium has been laid down; stringy if much fibrosis is present; or is no longer visible if absorption takes place. In describing the appearance of these shadows as seen in the active, healing and inactive cases, various terminologies have been used. Some observers have become accustomed to use the language of the pathologist and speak of the new or active lesion as "exudative" while others describe the areas as "soft" or cloudy. As healing progresses by calcification, fibrosis, or a combination, the shadows are spoken of as calcific, fibrotic, fibro-calcific if one uses the language of pathology, or simply as "hard," stringy or dense. At the moment there is no generally accepted or completely satisfactory terminology but this is less important than an understanding of the meaning behind the words. We are trying to classify with reasonable accuracy the potential safety or danger of the lesions into groups which are predominantly safe, unsafe, or doubtful as far as insurability is concerned.

Before interpreting any x-ray, however, it must be technically satisfactory and readable. If so, the first question is whether it shows an "Average Healthy Chest." If a lesion characteristic of tuberculosis is seen, it is classified by extent (Minimal, Moderately Advanced or Far Advanced) conforming to the criteria of the National Tuberculosis Association. Since our experience shows the group of persons whose disease has reached the Far Advanced stage are not insurable, extent

alone is enough for action in these cases. Otherwise the lesion is classified as healed, unhealed or doubtful. Only those films which show what is believed to be a healed or inactive lesion should be considered immediately for insurance, and even in this group it is not implied that there is no danger of reactivation. We expect, in fact, that the disease will reactivate in a small percentage of persons in this group and that they will die of tuberculosis, but this can be taken into account within limits of safe underwriting by classifying them as substandard risks.

Other factors which should be taken into consideration when dealing with an applicant of this type are the length of time during which the disease probably has been inactive or healed, the age, race, height and weight. Without going into much detail, the following principles were used:

1. Period of Inactivity or Healing of Lesion: If a history of cure is obtained, the longer the period of arrest, the safer and better the risk. We believe that a minimum period of five years of arrest is desirable.
2. Age: The younger ages require a slightly heavier rating.
3. Height and Weight: The applicant should not be too far removed from the average.

The results upon 1,817 lives are given without more discussion in the following table.

It is a pleasure to acknowledge the help of all those who have assisted me in this report. To Mr. G. D. Shellard, who made this paper possible by preparing the table, Mortality Experience, I am particularly grateful.

PRESIDENT JIMENIS—Thank you very much, Dr. Fellows and Dr. Reisner and Dr. Kiessling. Is there any discussion on this subject?

Mortality Experience of Insurance Applicants
Examined by X-Ray in the Home Office of the Metropolitan Life Insurance Co.

X-Ray Diagnosis	Ratio of Actual Deaths to Expected* Deaths	Total Actual Deaths	Ratio of Actual TB Deaths to Expected TB† Deaths	Actual TB Deaths	Number of Lives Exposed	Number of Life-Years Exposure
Far Advanced Tuberculosis	388%	14	3,333%	12	55	456
Active Tuberculosis (Not Far Advanced)	187%	14	824%	7	123	1,131
Healed Tuberculosis	79%	29	49%	2	568	5,240
Healed Primary Tuberculosis	58%	6	0%	0	198	1,830
Healed Pleurisy	86%	5	132%	1	108	1,030
Negative X-Ray (History of Lung Impairment)	89%	30	20%	1	765	7,101
Total		98		23	1,817	16,788

* Expected deaths based on Metropolitan's Ordinary (excluding $5,000 Whole Life) 1936–1940 experience.

† Expected deaths from tuberculosis based on average tuberculosis death rates for white males in the United States for the years 1931–1940.

Dr. Berthold T. D. Schwarz—Apparently you predicate your decision entirely upon the x-ray findings. Do you exclude entirely from consideration how long ago the history of active tuberculosis occurred?

Dr. Fellows—No. The experience with our Home Office employees, which has been entirely paralleled by that of the employees of the Prudential, is that we have never believed that is was safe to insure a case of tuberculosis within five years of a cure.

On a questionnaire which I sent out, I was amazed to find that over half of the companies considered two years discharge, not two years arrest. Some of the companies may have gone into the question of the condition at the time of examination, as well as at the time of discharge from the sanatorium, but this was not indicated on the questionnaires. Unless my memory plays me false, there are twenty-two companies which will accept, or at least consider, an applicant for insurance within two years of discharge from the sanatorium, assuming, I think, that the case has been arrested for two years. In our experience, and in the experience of the Prudential, two years is not nearly enough. A case of tuberculosis which was active, in our experience, should not be considered before five years have elapsed.

Dr. Hugh B. Campbell—After a number of years in sanatorium work, I think in view of the last question which Dr. Fellows answered, it might be well to keep in mind that the patient has the same frailties as many other patients.

If you get the history of a discharge a few years ago from a sanatorium, it does not mean necessarily that he was discharged by the medical authorities even as an arrested case. A great many people leave the sanatorium against medical advice. I can only say, from the experience which I have been fortunate enough to have had, that at least in the early years of a tuberculous case it is wise to adopt one of the dictums which Dr. Spiller used to give us down in Pennsylvania, on

syphilis, when he would say that the only thing certain about syphilis is its uncertainty. I think the advisability of considering an individual for insurance before his disease has been arrested for four or five years is highly questionable. I should say that that is the only basis upon which the individual should be considered for insurance.

I have wondered about this weight factor. In thirty years of experience in sanatoria, I think I have seen as many fat people die of tuberculosis as I have slender people. In other words, weight is not an essential. It is well, as Dr. Fellows said, to have the applicants as near average weight as possible, but I do not think that should be one of the chief influencing factors.

On the question of the x-ray diagnosis, I think even if you are accepting those after a five-year period, you can be safe only if you take the x-ray at the time the application is made, which is presumed to be five years after, and also have the opportunity to review films four, three, two, and one year after the patient is discharged. In that way you can observe the stability of the arrest of the tuberculous condition.

Dr. Arthur J. McGanity—I would like to ask, Mr. Chairman, what the definition of cure is? I do not know what it is in the United States, but our system, in the part of Canada in which I live, is to have a great many of these people discharged from sanatoria and then have them return from time to time for air or pneumothorax treatment.

Was Dr. Fellows' report based on those who have cessation of treatment? That is what I want to know. Have they had treatment after discharge from sanatoria, or have they had two years or five years of complete cessation of treatment and returned to civilian occupation of some sort?

Dr. Harold M. Frost—Dr. Fellows says that it is not safe to accept an individual until five years from arrest. Are you talking about standard insurance or substandard insur-

ance? If the latter, do you mean that you cannot rate an arrested case under five years heavily enough to be safe?

Dr. Charles A. Peters—There are large numbers of young fellows coming up for insurance now who were discovered to have a small lesion on x-ray examination for admission to the military. I am personally interested because my own son was one of them.

A great many of these boys' x-rays now are quite normal. Are those acceptable at normal rates a year after an x-ray is quite negative?

Dr. Schwarz—As a follow-through on the question that I asked, I believe it is very important that there should be a time test of the cure. But that factor is going to be a little more complicated as we go along and perhaps more favorably resolved by the use of streptomycin in certain selected cases.

I happen to know of at least one young man who had a cavity complicating active tuberculosis. The cavity cleared within a period of two weeks, and his x-rays, from the time he first showed clinical evidence of the illness, have been completely negative. For the first three months, the lesion on x-ray showed very rapid regression, and for a period following those three months the x-ray evidence has been nil, so much so that phthisiologists and roentgenologists who look at the x-ray are unable to find any evidence of tuberculosis in that chest unless they have the history given to them and some little assistance as to where they might look for a lesion.

Dr. Karl W. Anderson—After five years, if the x-ray indicates that the lesion has calcified, how soon would you consider them for standard insurance?

Dr. Fellows—First, I would like to express a word of thanks to Dr. Campbell. I was waiting to see if someone from the sanatorium might not give me an opportunity to make another good friend, because I make a lot of friends by disagreeing with them; but he was corroborating, as I take it, our opinion.

The question of weight is not too important as long as it does not deviate too greatly from average weight.

Just because a patient has left a sanatorium does not necessarily mean that he left either with the consent of the sanatorium authorities or that he should be considered as an arrested case.

If we can get a series of x-ray films, we consider them a great deal more help than a single film. However, we worked on whatever we had. Usually we do not get a series. I should think we did not have a series in one per cent of the cases. Usually it is a single film. The results that we showed were therefore based on a guess as to what was going to happen on a single film.

As to Dr. McGanity's question about what we call a cure, and whether these cases we are talking about are considered cured, none of the cases in this series, as far as I know, would be considered active. By that, I mean they were not receiving any active treatment, and furthermore, the average applicant for insurance who sends in an x-ray film, sends it in either as helpful evidence or because an x-ray has been taken in the course of a survey, or we take one in the Home Office. So the question of activity in any of the cases in this series does not involve an active case, as far as we know; and the applicants were not active as far as they knew. There is one notable exception to that. The most spectacular case of loss of memory was an applicant who came into the Home Office one day and applied for $10,000 worth of insurance. He had a pneumothorax on one side and an extensive lesion on the other. He said he had never had any disease and was perfectly well. He had forgotten that he was collecting disability from our Company.

Dr. Frost asked whether at the end of five years I believe we could consider an applicant either with or without rating? Your President said he would consider with rating after five years. However, we would not consider under five years in any event.

DR. FROST—How high do you rate?

PRESIDENT JIMENIS—It all depends. Our chart on this has two positions, one horizontal and one vertical. One is the relation of time to the disease, and the other is the extent of the disease at the time that he had it actively; the rating is based on a combination of those two factors. We do not ordinarily insure anyone who has had any activity or pneumothorax within five years. Now, there may be some exceptions to that. When you get a well authenticated history, you can make an exception, but the run-of-the-mill case gets no insurance prior to five years, and the amount of the rating starts high and is gradually reduced.

DR. FELLOWS—Dr. Anderson wanted an answer to the same question—what will you do with a healed minimal case with no history of treatment?

PRESIDENT JIMENIS—I assume you were speaking about those whose healed tuberculosis was discovered at the time of examination for the Army. For a while we were considerably worried about them, naturally. There is no question but that some of them do break down. But as time goes on we are becoming less and less interested in that history, and I think within a short time now we will ignore it. Do not forget that prior to the war we were insuring all of these people without the advantage of the Army examination screening.

DR. FROST—May I ask another question? I do not think I made my point clear. The point is this—why is it not possible, within the five-year cure, and you have already mentioned that twenty-one of these companies, and my company is one, issue substandard after two years' arrest—why is it not possible for them to apply a rating within that period to be safe?

DR. FELLOWS—I cannot answer your question from an insurance standpoint, but I can tell you what happened with six hundred of our Home Office employees, all minimal cases and asymptomatic, picked up purely by survey methods, cured

at Mt. McGregor where the length and conditions of cure would be considered ideal, and then returned to the job. There was in the first group a five per cent recurrent rate per year, ending between the fifth and sixth years. In other words, minimal cases which certainly would be considered ideal as far as treatment and cure are concerned, have had for the first six years a high recurrence rate, and at the end of the sixth year between 25 and 30 per cent of these cases had recurred. We translated that into an insurance action and a belief that if there is such a high chance of recurrence we could not take a chance where we knew the disease had once been active.

It may seem inconsistent, when we are willing to form an opinion on an x-ray film and consider the process healed. Dr. Reisner, I think, gave you the key to that when he pointed out, in these routine surveys, the many cases, which have a characteristic appearance detected by x-ray, who never knew they had been sick. When they had their disease, and it healed, they had enough resistance to heal spontaneously, or at least to cause it to become inactive without treatment. I think probably that is the answer to the question of why we can get a better result with those found on a survey than with those who have had known disease and were cured in a sanatorium. In cases with a history of active disease, knowing that within our group there has been a high recurrence rate we prefer to wait until the danger of recurrence is past. That may not be very scientific but it seems to work.

Dr. Frost—Your experience is based on recurrence rate, not actual mortality?

Dr. Fellows—Yes, it is recurrence, which means that we were dealing with an active disease; and not wishing to risk the hazards of active tuberculosis, we would like to be sure it is inactive or healed.

There was one other question on streptomycin. I had an opportunity last July to go to Chicago with five other people and go over some 1,800 to 2,000 cases of veterans treated with

streptomycin. It was a grueling experience. What they were trying to determine was the type of case which does well with streptomycin, the criteria for acceptance for treatment with streptomycin, and the results of treatment with streptomycin.

I do not know the results, and if I did I would not feel at liberty to tell you because there is some security involved. But we saw the most contrary experiences with streptomycin. It does queer things. If there is any logic as to the use of streptomycin, and there probably is, we do not know yet how to use it. We do not know how to select the cases, how much to administer, how long to give it, nor when to stop it.

I would like to ask Dr. Reisner if he would like to say anything about streptomycin.

Dr. Reisner—There is not very much I can tell you about streptomycin. I think Dr. Fellows probably knows more about it than I do, having attended the large meeting in Chicago; and we know the Veterans Administration has a very extensive stretomycin program now, probably more extensive than any other organization in this country.

My own personal experience with streptomycin is extremely limited, but with regard to that particular case the doctor has mentioned, let us hope that the beautiful effect has been accomplished with the streptomycin and that many others like that will follow.

As Dr. Fellows has indicated, we really do not yet know how to select the cases, although there is no question that the drug has a definite effect in some cases. Whether that effect is lasting is another question.

You are all acquainted, I am sure, through the reports and the literature, with the experiences with streptomycin therapy in acute tubercular meningitis which is almost always fatal, and in a certain proportion of the cases the disease has become arrested. Previously we did not know what it meant to arrest a case of acute tubercular meningitis. On the other hand, it

is only fair to say that those groups that have worked with the drug have, up to now, recorded a recurrence in a very large proportion of these cases of acute tubercular meningitis, although very encouraging initial results have been obtained.

A case such as the doctor has described where there was a cavity found on x-ray, with positive sputum, and following the administration of streptomycin there was complete disappearance of all the x-ray findings. We do see those cases happen without streptomycin. The only thing to do is, from now on, under such controlled conditions as the Veterans Administration and some other agencies have in mind, to accumulate sufficient experience on such cases with an apparently early type of disease, preferably before destruction of tissue has occurred, to show that such good results are accomplished considerably more often with the use of the drug than can be expected to occur spontaneously.

All I can say about streptomycin I have said, but while I am here I would like to say this, that the remark Dr. Campbell made is a very pertinent one, namely, before making a decision as to the status of tuberculous disease, one should make an attempt to obtain as much evidence as can be obtained on the basis of history and previous x-ray findings. Naturally, when we can do that we feel so much safer in rendering a decision, but at the same time we have to be realistic—and I am not speaking from the point of view of insurance, because I know nothing about insurance. The fact is that we are discovering daily thousands of cases with some tuberculous lesion of the lung, and we are making these discoveries accidentally. Not only have the Army and Navy done it on about 25,000,000 people, but civilian agencies, health departments throughout the country, the United States Public Health Service, and voluntary agencies are doing it constantly. In fact, it is becoming difficult to conceive how a person will become able in the near future to escape having a chest x-ray, and we believe everyone should have it. Therefore, it is to be expected that very large numbers of persons will be

discovered with such lesions without previous evidence of any sort, such as positive history, sputa, or previous x-ray findings.

We are not claiming, as Dr. Fellows I believe has emphasized, that the attempt to determine the importance of the given lesion on the x-ray is infallible—in fact it is fraught with many risks—but at the same time we have to come to the point of trying to get the most out of the available evidence.

PRESIDENT JIMENIS—Thank you, Dr. Reisner. Dr. Kiessling, would you like to add a word on this?

DR. KIESSLING—I would like to say something about selection. In the first place, we do not accept any application, for instance, with a personal history of tuberculosis without an x-ray, because we want to know what we are dealing with before we quote the rates.

We divide our cases into far advanced, moderately advanced, and minimal lesions. The far advanced we will not accept at all. The moderately advanced we accept after eight years. This is probably the most conservative attitude toward tuberculosis that any of the companies adopt, of all those represented here. We will not accept moderately advanced tuberculosis under eight years, and then they are considered no better than substandard special class C, carrying a mortality of about 175 per cent.

With the minimal lesions, we will not accept people with tuberculosis until they have been arrested for five years, and then they are no better than class A if they have a visible lesion on the x-ray. If their x-ray clears completely or shows just the faintest fibrotic strands, they eventually become standard after ten years.

DR. A. ALLISON WILLS, JR.—I do not wish to complicate this discussion by injecting differential diagnosis into the picture, but in reviewing x-rays of histoplasmosis how are we going

to differentiate that condition from calcified miliary tuberculosis, and how are we going to treat histoplasmosis as an insurance risk?

PRESIDENT JIMENIS—We will go to the roentgenologist for that.

DR. FELLOWS—Histoplasmosis carries some morbidity perhaps but probably no mortality at the time you see it. Then it is a question as to whether it is healed miliary tuberculosis or histoplasmosis. As a matter of fact, probably many of the cases we call healed primary tuberculosis may be histoplasmosis. Healed primary tuberculosis and histoplasmosis which has become rock-like in character, in our experience, give the same results in the long follow-up as the individual with the hospital x-ray of an average healthy chest.

We encountered that about seventeen years ago. We had applicants for employment who came to us with these rock-like things, and we did not know whether or not to accept them for employment. So we accepted them, and the reason we accepted them as applicants for employment was that cases of active tuberculosis did not become superimposed upon these hard, rock-like areas. But that was not the end of it. We took 500 employees with either histoplasmosis or healed primary complications, 3,000 employees who had nothing in their chest films, and we followed them for ten years. The percentage of development of pulmonary tuberculosis was identical in each group and negligible, so that from a practical standpoint I do not think it makes any difference whether they are histoplasmosis or healed primary tuberculosis.

There is an interesting sidelight to minimal active tuberculosis, in that it has a pretty characteristic appearance on a film, and some of the things that we think are active pulmonary tuberculosis actually turn out to be carcinoma. I would rather reject, I think, active tuberculosis and have it turn out to be carcinoma than not, and certainly I would rather refuse them than accept them on the basis of minimal

tuberculosis. In our series there were five or six which I called active minimal tuberculosis that on follow-up turned out to be carcinoma. By differential diagnosis these cannot be distinguished, but of course neither one is acceptable.

PRESIDENT JIMENIS — It is a pleasure now to welcome an old friend of this Association whose reputation reaches all parts of the world and who has never failed to bring us instructive and interesting information from one of our leading clinics.

Dr. Frank Howard Lahey's distinguished career has, among other things, included service to his country in two wars, the presidency of the American Medical Association, and at the present time he is Surgeon in Chief at the New England Baptist Hospital and Director of the Lahey Clinic in Boston. Dr. Lahey!

LESIONS OF TERMINAL ILEUM, COLON, AND RECTUM

FRANK H. LAHEY, M. D.

The Lahey Clinic, Boston, Massachusetts

I would like to speak to you about lesions of the rectum, the colon, and the terminal ileum. To be specific, I want to speak to you about these lesions because it seems to me that you are in a unique position to do so much about them. You are not only in a position to find carcinomas of the colon and rectum early, but you are seriously concerned, it seems to me, in your occupation and profession, with promoting earlier diagnoses of these lesions, and thus longer life.

With your examination of so many presumably well people in the field, it seems to me you are in the position of being able to do the thing that should be done, and that is improving even more the results which are now good in this field of surgery.

One could be quite complacent about these lesions, with the exception of one. When I speak of lesions of the colon, rectum, and terminal ileum, I would like to use the time particularly for carcinoma of the colon and rectum, ulcerative colitis, and so-called sclerosing enteritis, so often called regional enteritis or regional ileitis or terminal ileitis. I would like to bring to your attention these three lesions and our experience with them; but first, before showing any of the lantern slides, I would like perhaps to philosophize a little about this subject of carcinoma of the colon and rectum.

I have often said that these feature articles I see in the *Saturday Evening Post* and other journals, in the way of advertisements urging upon people better attention to some of the things which have to do with their health, are excellent. You have, through these, an opportunity to reach people that we as phy-

sicians do not have. I mean that the life insurance companies have this opportunity, but you and I as physicians could not do these things. I would love to write an article for the *Saturday Evening Post* about cancer of the colon and rectum, but I would do more harm than good, and I would do more harm than good in two ways. First of all, I might stimulate an undue fear, although I would not be so greatly disturbed about that. But it would be wrong for me to do that because it would also stimulate just criticism of myself and I would inevitably get bad publicity from it that would react against what I was seeking to do.

Why do I say this? The results in cancer of the rectum and colon are excellent. We have operated on approximately 3,000, probably a little more, of these patients, and 50 per cent of them are alive and well after five years, without recurrence. That is excellent, but if you break the cases down into groups in relation to late and early diagnosis, they get worse and worse.

In the clinic, Dr. Swinton took 100 cancers of the right colon, 100 cancers of the left colon, and 100 cancers of the rectum—proven by removal—and asked some simple questions such as, "Was there an alteration in bowel function? Was there a change in caliber of the stools? Was there obstructive type of pain?" When you realize the answer was "yes" in 97.7 per cent of the cases, you cannot avoid the implication that the diagnosis, or a very considerable suspicion of it, could have been made with the patient in Portland, Oregon, and the history shipped here to us to read. In other words, when out of these cases 97.7 per cent had the history of this possible lesion, the diagnosis should have been made earlier.

We have an operability rate of 92 per cent, but that is misleading. We can do the radical operation on 92 per cent of the patients who come to us. That is fine, except that they have been through two or three sieves before they come to us, and that is not comparable with the depressing figures that one sees in general hospitals and still more depressing

in rural communities. And so, I would like to see your companies promote a better publicity on the need for complete investigations, gastroenterologic, proctoscopic, and sigmoidoscopic, on the basis of some of these points demonstrated in the history.

There is still another thing it seems to me in which I would like to promote your interest. That is the lack of any question in the mind of anyone who lives with this subject, as we do, concerning the relationship of carcinoma of the colon and rectum to polyps and to adenomas. That involves the question of whether you have been fair to a patient after a complete physical examination, because he is placing false dependence on the thoroughness of your examination if he has not had a good proctoscopic and sigmoidoscopic examination included.

Probably if we speak of just a few things to call to your mind how close the relationship is between these precancerous lesions and later carcinomas of the colon and rectum, it will be helpful. Let us realize that 75 per cent of all the cancers of the colon are somewhere between the splenic flexure and the anus, and that 65 per cent of all the polyps are in the same region, again indicating the probable relationship between the two. Also, one must realize that all carcinomas of the rectum, except the occasional squamous cell ones, are adenocarcinomas or malignant adenomas; that again indicates the relationship between polyps and adenomas. Furthermore, if you live with these lesions as we do, you will be struck by the fact that at any level they are much alike. They have excavated centers and raised peripheral edges. They spread out similar to the burned out bed of a fire built in the grass. They show every evidence of central origin and circular peripheral spread. They are, in the beginning, all at a single point on a lateral wall, and as a result of this peripheral spread, eventually surround the lumen of the bowel to become annular and later to extend up and down and to canalize the bowel. I shall call your attention later to the relationship of the time period to

the development of these two lesions, annularity and canalization, as shown by the roentgenogram.

We feel very strongly that there is no physical examination today which is complete without a good proctoscopic, sigmoidoscopic, and a good contrast enema picture of the colon. The First National Bank in Boston became interested in annual examinations and sent us their top fifty executives, as have other companies interested in general physical examinations. It is interesting to realize that of the first twenty-one of these persons examined four had completely unsuspected polyps. It is probable that of all the general population, 5, 6, or 7 per cent have polyps.

Not all of these are going to become malignant, but a very definite percentage of them will become malignant. Twenty-five per cent of these will be multiple and perhaps only one may be found; if you fulgurate one, another one may become malignant. Nevertheless, each one found and destroyed is a possible patient saved from possible resection of his colon or an ultimate fatality from hopeless extension. It is for that reason that I am so glad to come here and talk to you about these lesions, to promote your interest in them and to promote a greater interest in publicizing some of the delicate aspects of this disease. We should have no false pride about discussing defecation, because the early diagnosis of these lesions is concerned, for practical purposes, with defecation and obstruction, and with change in quality and character of the stools as to diarrhea, alternating diarrhea and constipation, change in bowel habits or change in caliber of the stool.

With regard to the location of malignant lesions of the large bowel, in a study of 1,457 cases in which operation was performed at the Lahey Clinic (1936 through 1944), 75 per cent were found in the sigmoid, rectosigmoid and rectum. The distribution was as follows: cecum 6.52 per cent (95 lesions); ascending colon 3.30 per cent (48 lesions); hepatic flexure 2.94 per cent (43 lesions); splenic flexure 2.40 per cent (34 lesions); descending colon 3.97 per cent (58 lesions);

sigmoid 12.42 per cent (181 lesions); rectosigmoid 9.47 per cent (138 lesions); rectum 53.32 per cent (777 lesions), and anus 0.6 per cent (9 lesions). The figure increases from the splenic flexure to the anus.

In general, these are the things in the histories that one should always have in mind in carcinomas of the colon and rectum: bleeding in the rectum, usually bright blood, of course, because of the fact that the lesion is close to the anus; discomfort, tenesmus and change in bowel habit. In the colon there is bleeding from the splenic flexure down, but from there to the sigmoid it occurs less often, and in the right colon much less often because of the liquid character of the stools. Mucus, change in bowel habits, and anemias are particularly related to the ascending colon. No one has ever adequately explained why it is that malignant lesions of the ascending colon can produce such high degrees of anemia and still have the lesion operable. Again, it is impressive that the same degree of secondary anemia in a lesion of the right colon found in one of the left colon would indicate that the lesion was inoperable. Many times I have seen secondary anemias in patients with cancer of the right colon with hemoglobin of 30 per cent and the lesion still operable. With this true of the left colon, they would rarely be operable because this would represent their degree of cachexia from metastases that would make them inoperable, while in the right colon this situation is due to some other as yet unexplained factor, possibly the fact that the right colon is a fluid-absorbing segment of the colon and filled with organisms of high virulence and noxious toxins the absorption of which may produce the secondary anemia. The lesions, of course, do have a considerable area of ulceration through which absorption may occur.

It is to be noted that in 100 cancers of the left and right colon there was alteration in bowel function in 80 per cent of those cases, abdominal cramps or pain in 57 per cent, abnormal stools in 46 per cent, and there were no symptoms in only 2.3 per cent. Again, of these cases in which the lesion was

present and removable, the diagnosis was in the history, at least suggestively, in 97.7 per cent of cases.

Now I want to go to this thing that seems to me so important and in which we have tried over the years to interest doctors. We would like to interest physicians in doing more proctoscopic and sigmoidoscopic examinations. They are not difficult. The apparatus is not complicated. The vision is direct. The light is now good. The tables are excellent. One can today, with relatively little experience, if he is delicate and reasonably gentle, do proctoscopic examinations and sigmoidoscopic examinations. It is for this reason that I would like to get people who do complete physical examinations to include in them contrast enemas, proctoscopic and sigmoidoscopic examinations. There are only two or three things to be said concerning these. It is difficult to perforate the rectum with a proctoscope, although we have seen two or three that have been perforated. It requires a high degree of persistence, it seems to me, and an almost unbelievable roughness to push a proctoscope through the bowel wall of a conscious patient without his getting off the table and resisting it. I do not mean to be facetious, but I do not think it is a dangerous procedure.

There are two or three things that are necessary. One is a good table; the second is a good instrument with a good light that will work; and the third is proper preparation. To try to proctoscope patients who are inadequately prepared means failure to see anything. To try to proctoscope them in positions that are inadequate also means failure to see anything. But if they are given 2 ounces of castor oil and asked to take an irrigation, and then have another irrigation if possible before they are examined, and they are thoroughly cleaned out, almost anyone can successfully use the proctoscope.

The typical polyp is removed by sigmoid fulguration. That is the type of polyp seen in the sigmoid and colon in which the colon is opened and the mucosa is snared off at the base

with a good, wide margin for safety. I have in the hospital now a woman whom I examined by proctoscope again yesterday morning in order to be sure. The pathologic report on her had come back, "The body of this polyp is malignant." If the polyp has malignancy only in the body or in the tip and there is no involvement of the base, we would not do an abdomino-sacral removal, or a radical resection of the colon. The reason is that polyps with pedicles, not the broad-based-ones, have a blood supply of only one or two vessels and one or two lymphatics. The histology of the epithelium covering polyps is atypical anyway, and it is often, we believe, a microscopic diagnosis. Futhermore, the results are so satisfactory in carcinomas of the rectum, and it is possible by contrast enemas and by proctoscopic examination to keep these under observation, that we believe it is unjustifiable to do radical operations unless the base is involved and unless there is ulceration. It is not necessary, we believe, in these cases to do more than local removals and then to keep them under observation. If it were a broad-based polyp, with a malignancy reported at the base, we would then consider radical operation. If the polyp is situated above the level of the peritoneal fold, it should never be removed in your office or in a clinic. The patients should be sent to the hospital, because as the base is fulgurated off it may be fulgurated so close to the bowel wall that there can be a perforation. If they are in bed for two or three days afterwards, on sitting up, this will in most of the cases have sealed off and you will not have it to worry about. The vessel that supplies these polyps through the pedicle is a good-sized one, and if you try to fulgurate them or snare them off with a fulgurating snare, you may cut the vessel. Often it will bleed so profusely that you have great difficulty in keeping the blood away so that the base can be found and cauterized. It is for that reason that we do not snare these polyps off any more. That is the quick way to do it. If they are large, we fulgurate them quite thoroughly and send the patients home. At the end of two or three weeks, they have thrombosed, sloughed, and the small

bud at the pedicle can then often plainly be seen and the base thoroughly fulgurated; thus we do not have to deal with hemorrhage. If it is a large, broad-based polyp, we fulgurate part of it and send the patients home and let the vessels thrombose and let it slough, and then fulgurate some more until we have it thoroughly destroyed, and then the base is thoroughly fulgurated.

Everyone who operates on many cancers of the colon and rectum has been chagrined to take out the cancer, to make a colostomy, and at the end of three or four days have a polyp protrude out of the end of the colostomy. We have also been chagrined to remove one carcinoma and later find that we had left another one behind. Therefore, one must realize that at the time the carcinomas are removed, one must search for polyps and also for other possible carcinomas.

I wish to bring to you also our errors in connection with these polyps. Never operate on one of these patients on the basis of one x-ray finding. Mark the lesion with an arrow; send the patient home and have him return in a week or two for another roentgenogram, and be able to demonstrate the same shadow at the same level. Failure to do that has caused me to operate upon two patients in whom I could not find the polyp. In one, I am sure it was present because we have since taken it out; but had we followed these through and marked them out each time on the roentgenogram, we would not have made that error. Have the roentgenologist mark these by arrow; then repeat the observation and be sure that the shadow which is said to be the polyp is always at the same level.

Those of you who have done proctoscopies in days gone by have suffered the same difficulties that we have in trying to do the examination with the patients in the knee-chest position or in the lithotomy position. The only adequate positions for proctoscopy are those using the Haynes or Buie table. With the patient hung face down, the rectum hangs by its attachment to the levators. It means that one does

not have to blow the rectum up with gas. If the patients are in that position, a little air must be put in, but nowhere near as much as in the other positions. These are the tables which are so important, I think, to successful sigmoidoscopies and proctoscopies.

A problem frequently presented to us is the patient with an intestinal obstruction in the sigmoid. The question is, is it a carcinoma or it is due to diverticulitis? Diverticulitis, as you know, is common in this location, particularly the obstructive type, and because of that we have made mistakes both ways, as has everyone who deals with these in numbers. We have assumed that it was diverticulitis and found it to be carcinoma of the sigmoid combined with diverticulitis, and we have made the mistake the other way. In this situation, we recognize the value of contrast enemas, showing so well the diverticula and the mucosal pattern intact or absent.

With the bowel empty, one can see the still filled diverticula and frequently no evidences of an obstructing annular lesion of the type that distinquishes carcinoma. The important roentgenologic feature which differentiates obstruction from diverticulitis and that from malignancy is the retention of the mucosal pattern in the obstruction due to diverticulitis. In obstruction due to diverticulitis the rugae of the mucosal pattern are still demonstrable, and rarely will there be a carcinoma when that is true. We have dealt with many of these obstructive lesions of diverticulitis. We have had a good many of them to deal with because they had produced obstruction or had perforated into the bladder. Occasionally they perforate locally and produce abscesses, but if they become abscesses they can be drained. They often produce quite permanent external fistulas, and these can be closed ultimately only by resection of such lesions. In lesions of this type, we do a defunctionalizing colostomy in the hepatic flexure. We send the patient home for about three months by which time the inflammatory exudate has been absorbed, and the bladder with its healed perforation is no longer contaminated.

We then reopen the abdomen with the distal bowel completely defunctionalized, resect the segment involved, do an end-to-end anastomosis and wait three weeks. At the end of three weeks, we give a barium enema to demonstrate that the anastomosis is intact and will hold fluid, and then cut the spur and restore the fecal stream.

Carcinomas of the right colon rarely produce obstruction because the cecum is large and its contents are liquid; but when it is at the head of the cecum it can involve the ileocecal valve, and one can at that level have early obstructive symptoms.

There are certain lesions from which one can deduce the time period of their existence with reasonable accuracy. Let us say that my thumb and forefinger put together as a circle represent the lumen of the bowel. Then, a carcinoma starts at one point, we will say here, on the circle, it takes about six months for a carcinoma starting on one side of the wall to surround the bowel completely. So any cancer of the colon or rectum which is annular is a minimum of six months old. When, in addition, it has canalized, it is up to another six months old. And so we can say that the completely annular carcinomas have more chance of being inoperable than do those which involve only one part of the lateral wall; and that the ones that are annular and canalized have even a greater chance of being inoperable than do the ones which are only annular and the ones which involve only part of the wall. This very morning I operated upon a patient with one at the splenic flexure exactly like this and still completely operable. So one should not assume from what I say that this is necessarily so in all of these cases. All of these cases deserve investigation as to operability. There is still another point to note on roentgenograms, and that is the hooking demonstrable by x-ray that occurs in carcinoma of the colon. You will note in carcinomas which have become annular that there is a little hooking always on the proximal side particularly, which indicates a certain degree of intussuscep-

tion that differentiates an inflammatory lesion of the colon so often from a malignant one. An annular malignant lesion of the colon cuts off sharply so that the flexible bowel beyond it tends to intussuscept a little over it, and that produces the hooks as seen on the roentgenogram.

The rectal sigmoid region, as I have repeatedly said, is the level at which roentgenologists tend to make mistakes concerning the diagnosis because it is at this level that the barium-filled redundant loops of sigmoid can topple over the lesion. If the lesion is fixed, it is difficult to mobilize the loops so that it is not overshadowed by a barium-filled loop and so not demonstrated. All good roentgenologists have learned to put these patients in the Trendelenburg position and to manipulate their sigmoids in an endeavor to demonstrate lesions particularly at the rectal sigmoid level.

I would not want to finish this subject of carcinoma of the colon and rectum without urging upon every medical audience radicalness in approaching them. We have been very radical and aggressive in handling carcinoma of the low sigmoid and rectum, and for that reason we feel unsympathetic to the recent popularity of operative procedures which preserve the sphincter. We believe that if a patient has cancer of the rectum, the surgeon has only one obligation, and that he cannot take on more than one obligation, and that is to give the patient the best opportunity to remain alive and well. If he attempts to be aggressive and radical, and at the same time tries to preserve the sphincter, in my opinion there will be cases he cannot do, in which in so doing he will limit the aggressiveness of the approach.

I would like also to say—and I just wrote an article* on it, published in the July 1947 issue of the Clinic Bulletin, the title of which was "The Truth about Colostomy"—that there is a tremendous amount of misinformation about colostomy. Over the years, this misinformation has been characterized

* Lahey, F. H.: The Truth About Colostomy. Lahey Clin. Bull., 5:130–136, (July) 1947.

by a statement which is true of one type of colostomy. It has been said to be a living death, but most patients know about colostomy only when done as a palliative procedure. It is unfortunate that the patients who have a cancer of the rectum or sigmoid which is inoperable and have only a palliative colostomy are articulate, very articulate, and so are all their relatives, because the lesion is so bad and because they actually suffer very greatly. But the patient who has had his lesion removed and been well for years with a colostomy which gives him no trouble is secretive, and he is very inarticulate. He has a good colostomy that does not bother him, but he does not want to go around boasting about it. He will go to an individual who has to have an operation for cancer of the rectum and will gladly talk with him about how well he gets on with his colostomy, but he will not publicize it in defense of colostomy as to how well he gets on with it. It is for that reason, I believe, that we must distinguish between condemnation of colostomy in the patient on whom it has been done for palliation and the patient in whom the primary lesion has been removed because they are quite different situations.

Our colostomies bother so little that we do not let our patients wear bags. In the supply department of the Clinic, we do not sell bags. If we have a patient who has a bag, we try to take it away from him and tell him he has not learned how to handle his colostomy. They do not need to soil themselves if they will adhere to a reasonable diet and learn to constipate themselves and to substitute irrigation for defecation. With that, they wear only a small elastic belt, or little trunks, and a small piece of gauze over the colostomy. It is extremely important to differentiate the two kinds of colostomy.

I am sure that there are a great number of people with cancer of the rectum and colon who are being closed as inoperable who should not be closed. We have operated on over 100 patients on whom we have done combined abdominosacral removal of the rectum and total removal of the uterus,

tubes, ovaries, and part of the vagina. It sounds unreasonable, but one woman is alive over five years after removal of the tubes, the ovary, the uterus, the left ureter and an atrophied kidney. There was in this case involvement of the ureter by extension of some of the carcinoma close to the rectum producing malignant obstruction of the ureter and atrophy of the kidney. We remove the uterus in those cases in which the carcinomas occur at the rectosigmoid, at the reflection of the peritoneal fold, and lean against the back of the uterus with extension to the uterus by contact. That is not metastasis, but direct extension. We have in the same way resected the top of the bladder a number of times because a sigmoid carcinoma can topple on to the top of the bladder. One can easily resect the whole half of the bladder without any difficulty and turn it in and then remove the contact lesion. Again and again, we have seen patients who have been closed as inoperable because they had a contact involvement of a loop of jejunum or ileum which could easily have been resected. I saw a man just last week at a check-up examination in whom, seven years ago, all the perirenal fat of the left kidney was involved by malignancy around a carcinoma of the descending colon. I had to dissect his renal vessels and his renal pelvis and his ureter, but he is alive and well because it was only involvement of the lesion by direct extension and not by distant metastasis.

It is for this reason that we urge that these patients be differentiated into those who have hopeless distant metastasis and those who have only local extension. We will, in addition, remove cancers of the rectum and colon in any patient who has metastasis in his liver if the metastases are limited in number, because we know that with the lesion out he will live longer, he will die much more comfortably, and that it has produced much more satisfactory figures.

We followed our palliative colostomies done on patients who had not had the lesion removed, and the colostomies done on patients who had had the lesion removed. Those

who have had colostomies with the lesion left in lived an average of fourteen months; those who have colostomies with the lesion removed lived an average of twenty-five and a half months, that is with metastasis to the liver, and they died much more comfortably.

What are the end results in these cases? These are the figures that I said I would present to you with the hope that I could interest you in earlier diagnosis. Of the five-year nonrecurrence rates in the cases with no node involvement, no adjacent metastasis, no blood vessel invasion, 90 per cent are alive and well over five years with no recurrences. Carcinoma of the rectum has three favorable things about it. One is that it is a low-grade type of malignancy; second, it produces early symptoms and third, it is so located that wide and extensive removals can be carried out.

In Grade 2 with involvement only of adjacent lymph nodes, only 37 per cent are alive and well; with node invasion and adjacent structure invasion, 30 per cent. Here is the thing that makes us feel we should always do radical procedures, that is when there is blood vessel invasion—only 14 per cent of patients are alive and well over five years. Carcinoma of the rectum is not unlike cancer of the thyroid which has the peculiar property of growing into vessels. Therefore, to do radical removals we want the levators removed; also the veins about the lesions in the rectum should be widely removed not only to get the lesion and the nodes out but also all or as many of the adjacent vessels as possible. I think that is as much as I want to say. We do almost all of the carcinomas of the rectum, and colon, too, now in one stage. Mortality rates will interest you. Mortality rates for carcinoma of the colon are 2.3 per cent and carcinoma of the rectum 3.8 per cent, with an operability rate of 90.7 per cent.

Now I would like to present to you another lesion that it seems to me we must all be on the lookout for because it is such a serious lesion if it is permitted to go to an advanced stage. This is sclerosing enteritis or so-called regional enter-

itis. In the Clinic, we have done radical resections of the terminal ileum, or the jejunum for this disease, depending on where it was. Dr. Kiefer in the medical department has divided them into three very good clinical groups, and that again is related to the time period: Group 1, enteritis; Group 2, obstruction; Group 3, fistulas. If they are permitted to get into the late group, they then become almost hopeless from any point of view that we know of because they have skip areas of involvement in their small intestine, and to get it all out would require so extensive a procedure that it would not leave them with enough small intestine to nourish themselves and to continue an existence, or a reasonable existence. In the early stages, stage 1, they have abdominal distress, diarrhea, weight loss, and anemia. This lesion involves, contrary to ulcerative colitis, all of the coats of the small intestine; after the acute process comes the cicatrizing or organizing process, and in the second stage, at a later day, they have symptoms of partial obstruction, a palpable mass, and marked malnutrition.

Later, if these lesions are left in, there will be external fistulas, chronic sepsis and postoperative sinuses. There will be fistulas externally and fistulas internally into the bladder, the vagina in females, into the sigmoid and other loops of the intestine. This is the third or fistulous stage. So it is desirable not to treat this group of cases too long medically but to submit them, we believe, to radical removal of all of the involved area, together with that portion of the mesentery and nodes which are so often involved. This lesion is not known to be but probably is due to a virus type of infection. The diagnosis is often mistaken for that of appendicitis. Twenty-five per cent of these patients whom we have operated upon have had their appendix removed under the mistaken diagnosis of appendicitis. From our point of view, that is a good thing, even though the diagnosis was a mistake. These do not perforate early because all of the coats are involved. It is a relatively slow process, and if the appendix

is taken out first, they have time to progress from the first stage into the second stage where the process quiets down. Then the peritoneal cavity becomes vaccinated to the infection and the process becomes chronic rather than acute.

The typical roentgenologic picture is the so-called "string sign" first described by Cantor. It is the result of the cicatrization of the terminal ileum. In the typical late stage with multiple areas of the bowel involved, treatment really becomes an insurmountable problem. In cases with fistulas we frequently do a lateral anastomosis around the fistulous area, and wait up to a year, giving the patients penicillin and intestinal antiseptics until the sinuses have been closed or at least diminished, and then resect the lesion at a later date. In this late stage, with multiple skip areas in the bowel involved by the disease they are utterly hopeless cases. If such patients had been subjected to an early radical resection, they would have had a chance. We have no interest in the conservative surgical approaches to this disease, such as lateral anastomoses around these lesions. Our mortality in 100 cases treated radically by resection is 2 deaths, both with abscess, and we believe that to be as low as will be obtained by any of the conservative procedures and that the recurrence rate and need for secondary operations will be less with this radical approach.

One has to remember that not only does this process involve the terminal ileum, but it also involves the jejunum. Also, one does well to remember that to investigate these cases properly, not only must one look at the terminal ileum but also at the other segments of the bowel. If one segment is found involved, the search must be extended to all the other segments to make sure that there are no skip segments elsewhere. There is something to be said about the lesions that concerns only those who do the surgery for them, that is that these lesions involving the terminal ileum frequently run over onto the ascending colon. Therefore, removal of the ileum only is not enough. We think these patients should

have not only the terminal ileum removed but all of the ascending colon.

We think any patient who has had regional ileitis radically and successfully treated, who has not had a recurrence within five years, usually will not have one, but prior to that any of them may have recurrences. If they have radical removals, I think there is less chance of recurrence. Out of 100 resections, there have been something like 4 recurrences that have required secondary removals.

We come now to the other lesion I wish to speak of, ulcerative colitis. We have had about 700 patients with ulcerative colitis. It is a terrible disease. We do not know what its cause is except that it is probably a virus type of infection. It is not a sclerosing disease that involves all of the coats, but starts as a mucosal disease. I wrote an article* for "Surgery, Gynecology and Obstetrics," urging earlier ileostomy by which we might save the lives of more of these patients. We used to wait until everybody was agreed that if they did not have the ileostomy they would die. In 80 patients who reached that advanced stage in which the family, the family doctor, the gastroenterologist, and we as surgeons had agreed that the ileostomy was necessary, the mortality was 22 per cent. By earlier operation we can overcome this. We now do them much earlier and we have the mortality down to 2 per cent. Ten years ago, an ileostomy was a terrible thing for these reasons: it could not be controlled; there was no way of preventing the patients from soiling themselves and smelling. The fecal stream could never be restored because the operation was done so late. We now have means of improving this very much. There is a bag called the Koenig-Rutzen bag, made in Chicago by a man who has an ileostomy. It is a bag with a flange that is made to measure and fits snugly around the ileostomy so there is no skin unprotected. It has a shoulder flange which can be cemented by a special

* Lahey, F. H., Earlier Ileostomy in Severe Ulcerative Colitis. Surg., Gynec. and Obst., 85:230–232, (Aug.) 1947.

cement which is mostly latex, so that it will cling closely to the skin and is watertight. The bag has a spigot at the bottom. It has a belt around it. This is watertight and odortight. These patients can now wear this bag comfortably; they can play golf; they can go swimming; they do not have irritation of the skin, and it has made the life of the patient with an ileostomy heaven compared with the hell it was before.

In addition to that, we have done some prophylactic ileostomies on these patients. In the patients who have not done well medically, we have done an ileostomy with the idea that we will put the colon at rest temporarily and replace the ileostomy so that the fecal stream will be re-established if conditions later permit. These are limited to the early cases and to those patients who could fulfil the following three requirements: if after the ileostomy, haustrations returned completely in the section of the colon involved; if all symptoms disappeared, and if on proctoscopic examination the rectal mucosa was normal. If all of these have occurred, we have replaced the ileostomies after first warning the patients and their families that putting the fecal stream back might very well reawaken the disease. We have re-established about 15 of these. Of the 15, about 5 have had to have the ileostomies re-established because it has reawakened the disease. We have the Koenig-Rutzen bag, and we have the offer to the patient that he may get his ileostomy put back; and that has made it possible for us to urge early ileostomies and to get the patients at earlier stages.

The following measures constitute our management in acute fulminating cases and medical failures comprise about 60 per cent of cases of ulcerative colitis. What is the medical treatment? It is a bland diet plus typhoid inoculation. If they are having acute episodes, at times you can, by typhoid inoculations, bring about a remission. The things that bring about acute episodes in ulcerative colitis are anything that will produce an allergic reaction, emotional strains and,

particularly, infections such as colds and grippe. We put these patients on bland diets. We educate them to avoid things that we know tend to bring about the acute reactions. But if in spite of those the case continues to fulminate, it is a medical failure, and in those cases as they become more acutely involved, there frequently is massive hemorrhage, subacute perforations, fistulas, obstruction or polypoidosis and it is in these cases that we advise early ileostomy. If after that the colon becomes the rigid, lead-pipe colon, we advise colectomy; and if the rectum is involved, we advise colectomy plus abdominosacral removal.

In 80 per cent of the cases of ulcerative colitis, the rectum is involved. When the rectum is not involved there is an excellent opportunity to obtain something that is desirable in these cases; not an ileostomy, but a colostomy. So in these cases we advise a colostomy and then remove the rigid lead-pipe colon which is never going to function again. In this way, we hope to avoid extension of the disease and have a colostomy instead of an ileostomy, because the colostomy is so much easier to handle. The rigid lead-pipe colon has another feature to it which is the thing that I mentioned last in the indications for ileostomy. It is the degenerative polypoidosis. About 4 per cent of cases of ulcerative colitis have developed carcinoma which we think is in some measure related to chronic irritation constantly present in these constantly inflamed colons.

I believe that if we could submit the patients with ulcerative colitis to earlier ileostomy, and if we could get a wider knowledge of how comfortable these patients can be with a Koenig-Rutzen bag, the mortality and morbidity of this disease would be brought down tremendously.

PRESIDENT JIMENIS—Dr. Lahey, we are tremendously grateful to you. It is one of the most interesting papers I have ever listened to. Are there any questions from the floor? If not, I can only repeat my thanks to you, Dr. Lahey.

For many years a teacher of Forensic Medicine at New York University and at Cornell Medical School, Dr. Milton Helpern is also co-author of a standard textbook on Lethal Medicine and Toxicology, with which I think most of you are familiar. We will now hear his paper, "Sudden and Unexpected Natural Death." Dr. Helpern!

SUDDEN AND UNEXPECTED NATURAL DEATH*

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Many deaths from natural causes occur unexpectedly, suddenly, or under unusual or suspicious circumstances. The Medical Examiner's law in New York City[1] provides for the routine investigation by the Medical Examiner of all sudden and unusual, as well as obviously suspicious and violent deaths. The office of the Chief Medical Examiner in New York City, established by an act of the State Legislature in 1915, investigates approximately 16,000 deaths each year (about 20 per cent of all deaths). Of these, more than half are due to natural causes.[2] Other localities which have adopted a Medical Examiner's law similar to that operative in New York City are Essex County, New Jersey; Nassau County, New York; and the States of Maryland and Virginia. Although Massachusetts has the oldest Medical Examiner's system in the country, dating back to 1877, the law in that State provides for the investigation of deaths which are supposed to have resulted from violence, and autopsy in such cases can be performed only after written authorization has been obtained from the district attorney, mayor, or selectmen of the district, city, or town where the body is found.

Much of the content of this paper is based on a recent study by Helpern and Rabson[3] of 2030 cases of sudden and unexpected natural death. Necropsies were performed by the office of the Chief Medical Examiner in the Borough of Manhattan, New York City; and, as has already been pointed

* The lecture was illustrated by colored photographs of the lesions in cases of sudden natural death. Examples were shown of the more common and also of the rarer diseases causing sudden and unexpected natural deaths following the classification given in the tables.

out, it is better to emphasize the unexpected character of the natural deaths occurring in apparently healthy people, rather than the suddenness. In many instances, the individual does not die instantaneously or immediately, but may linger from several minutes up to twenty-four hours or more after the onset of the illness. The expression "natural death" implies that death was due entirely to disease and that traumatic injury or a poison did not play any part in bringing it about. It is sometimes difficult to draw a sharp line of distinction between natural death and death which may have been remotely initiated by some external factor. Thus, cirrhosis of the liver complicated by portal obstruction and a fatal hemorrhage from esophageal varices may be a sequel of infectious hepatitis or of syphilitic infection; but it may also result from chronic alcoholism or from acute or subacute liver necrosis produced by various poisons.

Sudden and unexpected natural deaths can be grouped into two categories:[4] The first comprises those cases in which death occurs in the presence of witnesses and under a variety of circumstances during which factors of physical and emotional strain may play a part. Death may occur during severe physical exertion or during preparation for a journey, during sexual excitement, or during or following an altercation. In other instances, it occurs without any obvious precipitating factors. The second category includes those cases in which the deceased is found dead under more or less suspicious circumstances. In such cases, the body may be fully clothed as though the deceased had been engaged in some activity. Or it may be found in bed, as though death had occurred during rest, sleep, or adventure. In such cases, there is the possibility that witnesses were present when death occurred, and perhaps responsible for it.

In the medicolegal investigation of sudden and unexpected deaths, the most important reason for the performance of an autopsy is to determine whether violence in any form—criminal, accidental, or suicidal—has been responsible. The cir-

cumstances under which death occurred, or under which the body is found, are important and may impel the performance of an autopsy. In many cases, the autopsy performed because of the unusual circumstances revealed that a death, seemingly natural and without external signs of injury, was the result of physical violence or of poisoning. The reverse is also encountered; deaths seemingly suspicious or violent because of circumstances or a superficial external injury were proved natural by necropsy.

The age of the deceased, reliable medical information as to past health, the presence of witnesses at the time of death, are factors that help to decide the necessity for autopsy as part of the investigation by the Medical Examiner.

Sudden natural death is less frequent in younger individuals, and when it occurs there is more often a suspicion of violent death leading to the performance of a necropsy. Sudden natural death of older persons is more frequent and more apt to occur under obviously nonsuspicious circumstances, so that investigation without autopsy is usually adequate. Objections to postmortem examination are more frequent in such cases, and the Medical Examiner ordinarily finds it difficult to justify its arbitrary performance. In some cases, the relatives desire to know the cause of death even though it is obviously natural, and necropsy is performed at their request.

Another reason for the performance of an autopsy is that an insurance claim or a civil suit for damage based on an alleged accidental injury is pending. Impending litigation or the prospect of the collection of accidental death benefits may prompt relatives to request autopsy, a procedure usually objected to when such considerations are not involved. In some instances, the existence of an insurance policy is concealed or denied for fear that an autopsy might disclose findings prejudicial to the beneficiary's interest. Determining the correct cause of death by necropsy may discourage unnecessary and unwarranted litigation. Without autopsy there

is too much room for speculation by misguided, interested, or unscrupulous experts.

Even when suspicion of foul play or trauma is absent, the insurance claim may instigate the performance of an autopsy which may reveal that death was caused by accidental injury, thus entitling the beneficiary to the accidental death benefit provisions of the policy. In other cases, where violence is suspected because of the circumstances, death is found to have resulted from natural causes. There are other more difficult cases in which trauma and natural disease processes have combined to cause death. When payment on an insurance policy is contested because of alleged misrepresentation in the application, the autopsy findings may clarify the point at issue. The presence of a fatal chronic natural disease does not, per se, prove that the deceased was aware of his condition.

In connection with workmen's compensation claims in which it is alleged that death was the result of or hastened by an accident arising out of or during the deceased's employment, it is to the interest of both the claimant and the insurance carrier, and most important for the referee who has to decide on the merits of the claim, to have the cause of death accurately determined. At the same time, there is an opportunity to evaluate the lesions found at autopsy with regard to their relationship to the alleged occupational accident or injury.

By careful and complete autopsy, the Medical Examiner contributes valuable data for the compilation of accurate vital statistics. He does this even though he is not concerned primarily with this problem as much as he is with the medical detection of violence as a cause of death. Of the large number of deaths occurring outside of hospitals, only those under the jurisdiction of the Medical Examiner come to autopsy. The Medical Examiner also functions as a public health officer by recognizing and first calling attention to early fatalities from epidemic disease.

The lesions which cause sudden and unexpected natural death, for the most part, fall into three categories.[4] The first and largest comprises natural disease processes of slow or insidious development that damage a vital organ without producing any serious symptoms until there is a sudden cessation of function. The most common example is sudden death from coronary artery disease. In the second category are the sudden and unexpected ruptures of blood vessels resulting in fatal hemorrhage. Examples are the bursting of an aortic aneurysm into the pericardial sac, or of an aneurysm of a cerebral artery resulting in fatal subarachnoid hemorrhage, or the rupture of an ectopic pregnancy bleeding into the peritoneal cavity. The third category includes latent or overwhelming infectious diseases which develop with varying rapidity without producing alarming or recognizable symptoms until death occurs. Examples are ambulatory lobar pneumonia and fulminating meningococcic infections. Of the three main groups of lesions, vascular rupture with hemorrhage is the easiest for the pathologist to recognize as the cause of death; infections are somewhat less obvious and the chronic degenerative diseases and neoplasms are most difficult to evaluate. In this last group there is always the possibility that a more subtle, immediate, not necessarily natural cause of death may be overlooked. For example, autopsy may disclose a degree of coronary arteriosclerosis sufficient to cause death, but dissection of the neck may reveal that the deceased died from a fracture of the spine and injury to the spinal cord, or from a foreign body impacted in the air passages, or from a lethal amount of poison as determined by chemical examination of the organs.

Pathologists who have had wide autopsy experience are well acquainted with the fact that chronic disease processes progress slowly, and that there may be little or no difference between the diseased organ at a period sometime prior to death and at the moment the diseased organ causes death. A degree of coronary arteriosclerosis or of cardiac hypertrophy may be responsible for sudden death of one individual,

while it may be encountered fortuitously in another in whom death was the result of traumatic injury, or poison, or some other natural cause such as a cerebral hemorrhage. A complete autopsy is necessary to exclude other more immediate conditions before ascribing death to a chronic disease process. The determination of the cause of death is basically an interpretive process which includes recognition of the pathologic changes found anatomically, bacteriologically, and chemically, and the selection of the lesion or lesions which were necessarily fatal at the time when and under the circumstances during which death occurred.

In the classification of causes of death, it is practicable to group them on the basis of pathologic anatomy and etiology, and to distribute them according to organ systems. Thus, the 2030 sudden natural deaths analyzed by Helpern and Rabson[3] when grouped according to organ systems gave results approximating those reported previously in similar studies by Weyrich[5] and by Lauren[6]. These are compared in Table I.

Table I

Distribution of Sudden and Unexpected Natural Deaths According to Organ Systems.[3]

	2,030 Autopsies (Borough of Manhattan) January 1937 to July 1943	Weyrich	Lauren
	Male 80.9% Female 19.1%	2,668 Cases Male 55.4% Female 44.6%	403 Cases Male 72% Female 28% (Includes alcoholism, excludes children under 14 years)
Heart and Aorta	44.9%	42%	51%
Respiratory System	23.1%	23%	12%
Brain and Meninges	17.9%	9%	15%
Digestive and Urogenital	9.7%	13%	9%
Miscellaneous	4.4%	13%	12%

Diseases of the heart and aorta lead with 44.9 per cent; the respiratory system follows with 23.2 per cent; the nervous system (brain and meninges) with 17.9 per cent; the digestive organs 6.5 per cent; and urinary tract 1.9 per cent; genital tract 1.3 per cent; the combined digestive and urogenital tract 9.7 per cent; a miscellaneous group totaled 4.4 per cent, of which three-fifths were represented by malaria artificially transmitted among drug addicts.

The further breakdown of the major categories of the 2,030 autopsies on cases of sudden death in the Manhattan series according to pathologic anatomy and etiology is shown in Table II.

Table II

Analysis of 2,030 Autopsied Cases of Sudden and Unexpected Natural Death.[3]

		Number	Percentage of Group	Percentage of Total (2,030)
Heart and Aorta 912 Cases (44.9%)	Coronary Artery Disease	617	67.7	30.4
	Syphilitic Aortitis	107	11.7	5.3
	Valvular Disease	83	9.2	4.1
	Cardiac Hypertrophy	35	3.8	1.7
	Spontaneous Rupture of Aorta	25	2.7	1.2
	Others	45	4.9	2.2
Respiratory 468 Cases (23.1%)	Lobar Pneumonia	176	37.6	8.7
	Bronchitis, Bronchopneumonia	133	28.4	6.5
	Pulmonary Tuberculosis	68	14.5	3.4
	Pulmonary Embolism and Infarction	31	6.7	1.6
	Others	60	12.8	2.9
Brain and Meninges 367 Cases (17.9%)	Cerebral Hemorrhage	110	30.4	5.4
	Subarachnoid Hemorrhage	93	25.7	4.6
	Cerebellar Hemorrhage	11	3.0	0.6
	Pontine Hemorrhage	11	3.0	0.6
	Cerebral Thrombosis and Embolism	27	7.5	1.3
	Meningitis	38	10.6	1.9
	Brain Tumor	29	8.0	1.4
	Others	43	11.8	2.1
Digestive and Urogenital		198		9.7
Miscellaneous		90		4.4

Two-thirds of the deaths referable to the heart and aorta were caused by coronary arteriosclerosis, its complications and sequelae, which is equivalent to 30.4 per cent of the total of 2,030 cases. This percentage would undoubtedly be higher if more of the older individuals who die suddenly were examined by autopsy. Syphilitic aortitis, complicated either by aneurysm, occlusion of coronary artery ostia, or aortic valvular insufficiency, accounted for 11.7 per cent of the group, or 5.3 per cent of the total. Non-luetic valvular disease is next in frequency (9.2 per cent and 4.1 per cent); then cardiac hypertrophy unassociated with valvular defects and in most cases secondary to arterial hypertension (3.8 per cent and 1.7 per cent); spontaneous rupture of the aorta (2.7 per cent and 1.2 per cent); the other unlisted causes in this group, including congenital heart disease, total 4.9 per cent and 2.2 per cent respectively.

Lobar pneumonia led in the respiratory group and was second only to coronary arteriosclerosis in frequency, comprising 8.7 per cent of all the sudden deaths. Next in frequency come bronchitis and bronchopneumonia (6.5 per cent), these deaths occurring chiefly in young infants; then pulmonary tuberculosis (3.4 per cent); and non-traumatic pulmonary embolism and infarction (1.6 per cent).

Among deaths from diseases of the brain and meninges, spontaneous cerebral hemorrhage, chiefly into the basal ganglia from the lenticulostriate arteries in hypertensive and arteriosclerotic individuals, was most frequent, comprising a third of this group and 5.4 per cent of the entire series. Subarachnoid hemorrhage, in most cases from the rupture of a cerebral arterial aneurysm, was almost as frequent with 4.6 per cent; included in this group are also some cases of intracerebral hemorrhage in locations other than the basal ganglia, localized subarachnoid hemorrhage overlying the insula, and occasional subdural hemorrhage, resulting from the rupture of demonstrable aneurysms of the cerebral arteries. Cerebellar hemorrhage and pontile hemorrhage contributed 0.6 per

cent each; cerebral thrombosis and embolism 1.3 per cent; suppurative meningitis 1.9 per cent and brain tumor 1.4 per cent.

The distribution of sudden and unexpected death by half decades according to organ systems in the Manhattan series revealed the greatest occurrence between 45 and 54 years of age; this was 10 to 15 years later than the percentage peak of population which was 35 to 39 years. Although males, according to the 1940 census, comprise 49 per cent of the population of Manhattan, sudden and unexpected natural deaths occurred four times more frequently among them than among women. (A similar preponderance of the male sex is found in the incidence of violent deaths.)

Racial incidence of sudden natural deaths was in proportion to population. White persons make up 83.5 per cent of the inhabitants of Manhattan and furnish 84.2 per cent of the cases of sudden natural death studied by autopsy. Negroes comprise 15.8 per cent of the population and yielded 15.2 per cent of the natural deaths. Negresses, however, contributed more than 26 per cent of the female sudden deaths, or almost twice the expected proportion.

It should also be borne in mind that the population of Manhattan is predominantly adult; 75 per cent are over 21 years of age.

Sudden Natural Deaths From Coronary Artery Disease

There is much misunderstanding of the symptomatology and manner of death in relation to the fatal lesions of coronary arteriosclerosis and this has been responsible for medicolegal confusion. The misunderstanding may be attributed in part to the lack of appropriate data from mediocolegal sources, but a share in the blame belongs to clinicians who attempt to evaluate coronary artery disease where death has been sudden without the usual clinical symptoms, on the basis of experience with the more common examples encountered in office and hospital practice. Pathologists, like the clini-

cians, have also analyzed their material with criteria gained from patients dying after days or weeks of illness. The problems confronting the Medical Examiner in sudden and unexpected natural death, of whatever cause, require standards of evaluation with which the clinician and hospital pathologist are not always familiar.

The finding of a severe grade of sclerosis of the coronary arteries does not, per se, mean that such disease is the cause of death. Death should not be attributed to disease of the coronary arteries without a complete study of the circumstances and a full autopsy, including examination of the brain and, where necessary, chemical examination of the organs. In the absence of significant change in organs other than the heart, with chemical findings unrelated to the death and with a review of the manner of death, death may be attributed to coronary artery disease when such disease is present in adequate degree.

Coronary artery disease as a cause of sudden death is almost exclusively a malady of white men. Only 6 per cent of the deaths from this disease occurred among women, all of whom were white. Three and seven-tenths per cent of the deaths from this disease were in male negroes, and there was only one Chinese, also a male. Thus, white men contributed 90 per cent of the sudden deaths from coronary artery disease. Although negroes contribute a very small share of the sudden deaths from coronary arteriosclerosis, far less than their percentage in the population, they contribute an excess number of sudden deaths from syphilitic aortitis with obstruction of the coronary artery ostia.

Three-fourths of the 617 cases of fatal coronary arteriosclerosis had no grossly fresh thrombosis of the vessels demonstrable at autopsy. In most cases, atheromatosis, with or without calcification, had produced significant diminution of the caliber of the lumen so that, physiologically as well as anatomically, the effect was not different from that of old

thrombosis. Functionally, a narrowed, diseased, sclerotic artery, even without complete occlusion, may be unable to furnish an adequate blood supply to the myocardium.

Non-thrombotic sclerosis was not accompanied by myocardial fibrosis in 45 per cent of the cases, and was accompanied by fibrosis in 50 per cent, while infarction was observed in only 5 per cent of these cases.

In the remaining 25 per cent of sudden deaths from coronary arteriosclerosis, the sclerosis was complicated by fresh or recent thrombosis, and of these 75 per cent had myocardial infarction with or without antecedent fibrosis in about equal proportions. A small number of deaths resulted from the rupture of fresh myocardial infarcts producing intrapericardial hemorrhage.

Sudden deaths from coronary arteriosclerosis begin to appear in the third decade and then to increase in frequency through the decades until the peak incidence is reached between 50 and 54 years. The incidence then gradually falls but its percentage incidence remains in excess of the percentage of the respective age group in the population.

Among the sudden deaths from coronary artery disease, every profession and trade was represented, with the more humble occupations predominating. In almost half the cases, death occurred in the street, during work, or in a public place; in an equally large proportion it occurred in the home or in a hotel room. At home, death occurred as often in bed as in all other parts of the household combined. Persons who died in hotels were most often found dead in bed although not always following a night of rest and relaxation.

As for the time interval between the onset of symptoms and death, this could not be determined in about a third of the cases found dead in bed. Of the remaining two-thirds, about 80 per cent died virtually instantly. Acute alcoholism does not play any obvious role in promoting death from coronary arteriosclerosis. In 80 per cent of a group of cases in

which chemical tests were made on the liver and brain, no alcohol was found. Of the remainder, one-third gave results of 1 plus or a trace.

Fifty-six per cent of the sudden deaths from coronary arteriosclerosis during a two year period had hearts weighing more than 400 grams. The hypertrophy was more marked in the fibrotic hearts, and more than half of the enlarged hearts were heavier than 500 grams. It was not possible to determine whether hypertension was a factor in causing the hypertrophy.

Spontaneous Subarachnoid Hemorrhage

Sudden natural death from this cause will be mentioned only briefly. Of the total of 2,030 cases of sudden natural death, there were 95 cases of spontaneous subarachnoid hemorrhage, or 4.7 per cent of the total. There were 59 men and 36 women including 22 negroes, 12 men and 10 women. The percentage incidence of women dying of this disease is therefore greater than their contribution of 19.1 per cent of all sudden natural deaths.

Deaths from this disease, with the few exceptions in the earlier years of life, occur in the years between 20 and 70 and most commonly in the years following full maturity and in middle age.

In most instances, the source of the fatal subarachnoid hemorrhage is a ruptured aneurysm of a cerebral artery. In two-thirds of the cases studied the aneurysm was identified, and in almost a fourth of the cases the exact source of the hemorrhage could not be located. In about 75 per cent, the aneurysm was on the anterior and middle cerebral and anterior communicating arteries.

Anatomically, the most common type of hemorrhage is the diffuse subarachnoid bleeding. Less often there is produced a localized hematoma over the insula, or extension into the subdural space. Spontaneous subdural hemorrhage from the rupture of an aneurysm of a cerebral artery can occur without

associated subarachnoid hemorrhage. In some instances the aneurysm is embedded during development in the adjacent cerebral substance; when such an aneurysm ruptures, intracerebral hemorrhage may result usually with, but at times without, extension into the subarachnoid space and ventricular system. There may be confusing premonitory symptoms without immediate loss of consciousness in such cases.

The individuals who died were engaged in all occupations. Death or collapse occurred in the home in 45 per cent of the cases and of these less than one-fourth were in bed; in the street or park in about 15 per cent. The exact circumstances immediately preceding death or collapse were not known in the two-thirds of the 95 cases occurring at home, in strange beds, or on the street. Alcohol determination carried out on 45 per cent of the cases revealed positive findings in 60 per cent of the cases tested.

Dissection of the cerebral blood vessels should be done as soon as the brain is removed from the skull, while the clot is soft and easily removed by washing. This procedure is especially important in cases of suspected criminal violence in which a suspect has been taken into custody pending the outcome of the autopsy.

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PRESIDENT JIMENIS—Dr. Helpern has earned our gratitude. That was a most constructive and interesting paper.

Gentlemen, our next presentation is a forum on Claims. Please remember that our moderator will be very glad to receive questions, written questions, from the floor, which can be passed up here so that he can have them answered.

Dr. Arthur J. McGanity is particularly well known to his Canadian confreres and very well known to us, too. It gives me great pleasure to introduce our moderator, Dr. McGanity.

OPEN FORUM
on
CLAIMS

ARTHUR J. McGANITY, M. D., Moderator

The Dominion Life Assurance Company

I consider it a great honor to have been chosen as the moderator of this session. In Canada we have been rather impressed by these forums as they have occurred in the past ten years or so. With our Canadian group we find that they are probably a little more relevant to our work up there than they are to a great group like this. We have found it expedient and, I think, well worth while to have started a sort of open forum of our own, of which we held a session last April with practically every member of the medical directors group in Canada present. There are many subjects that come up in the course of a year on which I am sure many of the men, particularly in smaller companies, would like to have a little guidance. We would like to have a little comfort from the experience of the greater companies, and we find that in an open forum, such as this, there is an opportunity of obtaining just that sort of information and that help.

I have glibly accepted the position of moderator without realizing that there were some very definite obligations in connection with the program. I want to say my word of thanks to your program committee for having arranged this program.

Now, the first speaker on our forum in connection with Claims is Dr. Ralph M. Filson, Associate Medical Director of the Travelers Insurance Company, who is going to talk to us on "Concepts of Claim and Medical Department Relationships and Co-operative Endeavors". I take pleasure in introducing to you Dr. Filson of the Travelers Insurance Company. Dr. Filson!

DR. RALPH M. FILSON—Mr. Moderator, Members of the Association, and Guests: One of the chief purposes of this forum is that of stimulating thought and discussion concerning the subject which has been assigned. I propose to offer some general observations and suggestions concerning what I consider to be a lack of balance in the attention and effort devoted by Medical Directors to ways and means for providing informed and practical medical advisory services to their Claim Departments.

I take the position that there has been and still is such a lack, on the strength of an extensive personal experience with claim medical problems as they have developed in branch offices and the Home Office of my own company. I have also failed to recognize any maintained attempt at formal liaison with insurance claim organizations on the part of this and other organized bodies in insurance medicine. I have likewise noted the comparative or almost complete absence of Medical Director representation in the councils and at the annual meetings of the International Claim Association.

Claims, after all, are the fruits of our underwriting. It is not my intention to have anything to say about those final fruits classified as death claims except perhaps to briefly mention the death claim which develops during the contestable period. There are, however, a great volume of claims occurring during the lifetime of individuals who are insured. The healthy interest of the membership of this body in the tree of insurance protection ought to be paralleled by its recognition of the need for better informed and more active and enthusiastic medical assistance in dealing with problems produced through the fruits of that tree. Is it not reasonable and equitable that while giving to medical selection its full share of medical departmental talent and experience in cooperative endeavors with company underwriters, we also accept it as our responsibility to provide through medical departmental channels something more than casual assistance to our claim executives? Medical problems, widely varied and

complex, exist to an equal if not greater degree in the claim field, and I am satisfied that members of our Claim Departments will wholeheartedly welcome additional Medical Department aid in their evaluation and solution. This will be particularly true if that aid is thoughtfully designed and competently rendered.

As a measure of the possibilities for illegitimate assaults against our insurance claim outlet, one has only to recall that a very few years ago it attracted, through its yield of profits, a carefully planned and organized attack by a group of racketeers. Many of these were members of honored professions who, in addition to keeping the claimant members of the gang well paid, lined their own pockets at a substantial expense to insurance companies.

An indication of the importance of vigilance in utilizing all available facilities to process claims so that they may be properly classified as regards merit and duration may appear in the following. During the year 1946 the company which I represent paid and allowed to living policyholders under Life Disability—Commercial Accident and Health—and the several lines of Group coverage a total of approximately $26,600,000. Now, if one recognizes the existence of a field within his company for more active aid on the part of the Medical Director, what can he suggest as bases for effectively dealing with this situation? Generally speaking, there should be, on his part, a thoughtful appraisal of those broader reasons for becoming more actively interested in the medical questions and problems arising out of claims. He may give consideration to these from standpoints of his company and the industry as a whole—the public interest—the inevitable part played in claims by his professional colleagues, and methods through which he and his associates can best contribute.

If a Medical Director approaches the subject from these points of view, I believe he will encounter some thought provoking material which will be likely to include among other features some of the following points.

1. While often in the purchase of protection the applicant may have bought on the basis of his relationships with a known agent rather than from an appraisal of the company represented by the agent, when a claim arises, he then measures the company and not the agent.

2. When Medical Directors define requirements which represent the basis for underwriting consideration, the applicant may decide that he will not be governed by those decrees.

3. The claimant appears in a different role under the terms and implications of a contract. These circumstances place the Medical Director in an altogether different position by requiring the claimant's co-operation in determining the facts while directing the method of their procurement to the mutual interest of the claimant and of the company.

4. Contrary to the applicant whose medical picture comes largely from company examiners, the claimant presents his own case. He either has or thinks he has a compensable loss as a result of injury, disease, or a combination of the two. He is maintaining probably that some impairing defect is of traumatic rather than non-traumatic origin. He is contending regarding the degree or duration of disabling influences from an accident or illness. He has had surgery which has to be classified for the purpose of determining the proper allowance. He is seeking financial benefits, often in sizable amounts, and in every instance he has to have support for his claim coming from a medical source.

5. Attending physicians who complete medical claim proofs bring the medical profession at large into a field of close relationship with insurance companies. This relationship is an important one and can be best understood and most fruitfully developed under guidance through medical departmental channels.

6. Statements by attending physicians, while constituting the framework of the eventual claim structure, are often completed without the doctor properly appreciating what may

be involved or the nature of his position in the picture. Frequently these statements are sketchy in content, lacking in details of history or of symptoms, and still more often in any objective findings to justify the diagnosis or the stated claim opinion.

7. Many attending physicians do not feel that it is a responsibility of theirs to refuse certification requested by a patient, or to devote the time necessary to explain why they cannot agree with the patient's point of view regarding cause and effect, or the disabling influence dependent upon an existing disorder. They conclude, and possibly not without a good deal of justification, that it is up to the insurance companies' representatives to interpret the contract under which claim is being made and to develop such detail as may be required to permit a decision regarding the merit of an individual claim.

Assuming the justification for medical departmental participation in the problems presented through claims, it is my concept that it is distinctly the duty and the responsibility of the Medical Director to establish the criteria for the use of Claim Department personnel in the evaluation of medical sections of claim proofs and in the recognition of either inadequacies or defects in those proofs. I suggest further that Medical Directors should make it their business to perfect, through a thorough review with Claim Department heads of their respective companies, those policies which are in effect for selection and reference to the Medical Department claims having a confused or complicated character along lines such as the following:

1. Contestable period death losses where through claim proofs or preliminary investigations there may be either evidence or suggestion of earlier histories which had not been disclosed at the time of underwriting.

2. Cases where alleged effects from stated causes are not recognized as usual or expected, or where factors other than

the stated cause appear to have had a role of responsibility in the production of the claimed effect.

3. Cases where either diagnostic detail or its sources are questionable.

4. Cases whose technical reports are difficult or impossible of proper evaluation by laymen.

5. Cases in which questions arise as regards character and extent of medical examinations to be undertaken on behalf of the company. These and many other parallel and related matters can only be satisfactorily evaluated and explained by qualified members of our Medical Departments. It follows also that the claim executive needs a thorough understanding of the medical aspects of these types of claims before he can administer them effectively, equitably or intelligently.

Cases involving any question which requires reference to a Medical Department from a Claim Department should be received and dealt with in the Medical Department with the same scrupulous care and attention as is given to the problems of medical selection in underwriting. Referred claim matters are never amenable to a simple "Reject" or "Do not Consider" course of action. They cannot be filed away because data are incomplete. There must always be an appraisal of available data however incomplete these are, and if additional are needed these must not only be fully defined but there must be an indication of the best source through which to seek them. When in any such case after it has been fully developed there is a decision that merit is not established, there must be a synchronous and parallel determination of the means and material through which such a decision can, if necessary, be defended.

Claim medical advisory undertakings call for certain attributes on the part of those who handle them most successfully. These, while required in all medical departmental assignments, are perhaps most necessary in dealing with the

type of medical problem which occurs in claim cases. There is a call for an especial interest in patient analyses of sets of medical facts or allegations, together with the ability to segregate what is often a small kernel of controlling truth after having sifted out much inconsequential or chaffy material. There must be sufficient power of imagination to allow consideration of possibilities apart from those appearing on the surface. There must be the ability to identify cases in which the sources of the claimant's medical proof are inadequate and to select sources through which defined additional data must be procured. There must be not only a willingness but a desire to pursue through proper channels any and all controversial medical matters until these are satisfactorily clarified. There should be a spirit of friendly and co-operative understanding with Claim Department personnel which will enable the doctor in appropriate cases to specify purposes and details of investigations conducted through other than medical channels.

Over and above all of these attributes, a Claim Department medical advisor must have, or be able to develop, the ability to reduce complex medical situations into terms understandable to his non-medical associates. I might add also that a doctor in this role should be willing to acquire at least a basic degree of knowledge regarding legal machinery and its operation, especially in connection with matters of evidence, its admissibility and its comparative value. In this connection, it must be remembered that in spite of all efforts to avoid litigation, it still confronts us occasionally in connection with claims, and when this means of resolving a dispute has to be employed it very frequently entails the introduction of medical testimony. Coupled with such qualities as I have specified, there is also the question of the part played by a Medical Director in selecting from among members of his department those to whom claim medical advisory duties are to be assigned.

These remarks constitute only the barest outline of views

regarding a field of fruitful interdepartmental co-operation. If they awaken an interest in more active medical departmental exploration of that field, and the possibilities of more effective service within it, their presentation here will have had value.

DR. MCGANITY—We are surely very happy to have heard this paper from Dr. Filson. I am going to ask if there are any questions now in connection with this paper, and we will ask Dr. Filson to answer them as they come along.

If there are no comments, I am going to ask our next speaker, Mr. Ralph Heller, General Manager of Claims of The Prudential Insurance Company, to speak to us on "The Medical Needs of the Claims Department." Mr. Heller!

MR. RALPH T. HELLER—I am very happy to be here with you this morning, though I must confess to have experienced a little surprise when I was asked as a claims man to appear on this program. I did not know whether it indicated that you were perhaps a little more democratic and less exclusive an organization than I had imagined, or whether I should charge it off to just another error in selection.

I want to talk this morning about the medical needs of the Claims Department, some of the specific types of service that we claims men would like to have rendered to us by men in the Medical Department, and to make a few suggestions that occur to me as to ways and means by which this joint medical-claim effort can be carried on with more productive results than has been the case in some instances hitherto.

Naturally, what I have got to say is conditioned by the circumstances under which I work and the background that I have acquired in my company, so that some of what I say would have little application elsewhere, or would obviously require modification. I am in a group that is set up as a non-medical claim department. We have a sprinkling of men with medical training in the group, but the rest of the men are without formal legal or medical training. We do have

an advisory medical section in the Medical Department and we have a litigation section in our Law Department which devotes itself entirely to claim matters. We make free and ready reference to the claim section of the Medical Department. Their judgments are usually determinative in what are purely medical questions, but their relation to us is primarily advisory.

A few words as to the situation out of which this need for medical help arises in the Claims Departments of the several companies: Judging by our experience, I should imagine that last year over several millions of claims of all kinds were paid. I obtained some figures from the Life Insurance Association which indicated that the total payments for all types of claim maturities was over $2,000,000,000 last year. Many of those cases permitted easy and ready disposition but there were tens of thousands of cases with medical angles of one kind or another that needed some sort of attention along medical lines prior to disposition.

There may be some difference of opinion as to how exacting some of the claim work may be, but there cannot be any doubt that in dealing with these many policyholders and claimants the reputation of the company and the general regard with which it will be held are determined by how effectively claim questions are answered.

Take, for example, medical questions—if in the Claims Departments of these several companies we have the facilities for providing an answer, or we have a means of reference to the claims section of the Medical Department, we can dispose of these cases with some measure of success. But, if we have not in our own divisions or in the medical services provided by the company the wherewithal to provide a sound and satisfactory answer to these questions arising on many different types of cases—additional indemnity cases, disability cases, group insurance cases, short-term claims—then obviously, in many instances we are going to render to the policyholders a service that is less than satisfactory. In other

words, we are going to provide a service which leaves something to be desired so far as the needs of the company are concerned.

Now, I would like to consider just how much medical knowledge we should attempt to provide for the Claims Department members, how much they can absorb and how this knowledge should be given to them. That is a proposition that is much easier to state than to answer, but, obviously, the difficulty in answering it does not do away with the need for an answer.

There are widely differing viewpoints as to just how much medical information we should attempt to convey to claim men, laymen without formal training, and how much we can expect them to profitably utilize. Some people would feel that but very little can be extended to them. In other cases, there would be arguments in favor of a wide extension of the privilege of using medical information. I think perhaps a reasonable viewpoint, and certainly a practical viewpoint—because we must all do and deal with our organizations as they are actually set up—would be this: to look forward to the communication of medical information to the Claims Department staff up to the full point to which their ability enables them to satisfactorily digest it and intelligently use it. I understand that in many instances that would not give one too much to work with.

Now, let us talk about the sources of the claim man's knowledge. How are we going to build up his knowledge in specific matters? I think that as an illustration I might use the disability claims file of the companies, which is perhaps the most outstanding example of the need for medical information. The claims man has available to him, first of all, the file of cases that he handles, and the value of this is not to be underestimated. If the man handles hundreds of cases, or thousands of cases through the course of the years, if he studies reports from attending physicians and from examiners, if he sees these cases terminate in recovery or death, if he

sees these cases and the medical findings collated and compared with the physical activity of the person involved, he builds up a sizable amount of valuable knowledge.

His next great source of information must be derived from the Medical Department itself on cases sent to the medical division, and, obviously, there will be many of these also. I think one of the most interesting things in Dr. Filson's paper was an attempt to set up some sort of a criterion, some sort of standards by which we could judge the cases that might properly be referred to the Medical Department. In other words, to set up some measuring rod by which the claim man might recognize that he is out of his department, technically speaking, and seek at that point the medical aid that is necessary.

These that are referred—they will be disability cases, cases involving laboratory data, many cases of sudden death, cases involving some measure of research, some of the alcoholic problems, some of the drug cases—afford a valuable and substantial basis for increasing the knowledge of the claim man relating to medical work. Obviously, these will not be enough. Much of that is too scattered; too much of it pertains to the individual case. Much of it lacks the basic background that is necessary to a proper understanding of the medical problems. I think this further need can be met to a considerable extent by the preparation, on the part of the interested medical men, qualified to deal in the claim field, of lectures and discussions relating to some of the basic medical problems. I think we should go through the body, system by system, with these laymen, give them the anatomy of the body, review with them the physiology of the organs, and talk with them about the signs of disease applicable to the parts under discussion. Take, for instance, the cardiovascular system. It would help if a qualified person lectured to them on the heart and blood vessels, how they operate; describe the signs of early stages of illness, the signs of serious illness, the signs that are indicative of necessarily disabling illness and of approaching dissolution; talk to them about how these things

must be interpreted and how they apply in terms of the physical activity of the insured. There will be some activities that are thoroughly consistent with the continuance of gainful work. Other activities will be plainly barred by the degree of disability described, and in other situations there will be present that degree of disability which would be perfectly consonant with either disability or with continued work, dependent upon the other circumstances obtained. Better still, if we follow up this kind of information by courses of training provided for laymen, we are then giving them something definite against which they can measure with some degree of accuracy reports that come in from attending physicians and examiners.

Now, I have referred to the disability field. Without enlarging on it much, the same thing could be said about the group surgical work, or group casualty work which has undergone a tremendous expansion in recent days. It would be quite foolhardy to attempt to administer the surgical schedule, particularly that portion of it which is related to the unscheduled operations, without some kind of knowledge as to the anatomy of the organs operated upon, and without some idea as to the nature of the operations. The men in our own company have performed this type of service on a number of occasions through the years, and we have found it exceedingly helpful in the training of our staff. I shall mention a few things relating to some of the practical sides of the work that tend to make it more effective. Some of this might sound a little petty, like captious criticism, but it is not intended to be such. The purpose in citing these things is this: if you do a fine job and put a lot of time and effort into training men in medical fields, you do not want that nullified or lost by failure to get it over in the best possible fashion.

We have been talking a lot about the claim men. The medical man needs some training in this field, too. Obviously, there is a good deal more to certain claims than medicine. Just as in many instances the medical facts are completely

determinative in the action to be taken, there are others in which the medical facts have little to do with the question of whether or not the company has a liability. Therefore, if a medical man is to do a satisfactory job, if he is to help the claims division in a practical way, he must make the effort to master something of the distinctively lay features of the claim. Secondly, provision has to be made to devote adequate time to this subject. This is not something that can be improvised or dashed off, nor the best results secured if today one doctor handles a case mixed in with new business selection problems, and tomorrow another one picks it up and renders another decision. Obviously, there is need for an important systematic and definite approach to the problem, and where the circumstances permit there is need for full-time service in this field.

Again, we need the decisions and viewpoints advanced to us in language that we can understand. Now, we do not ask you to get down completely to our level, but we would like to have a pretty clear idea of what you are discussing. There is need for oral discussion. Much of the work can be handled on a written basis, but there are many of these problems in which oral discussion is necessary if the man who is going to render the decision on the case is to have a complete and satisfactory understanding of his problem. Moreover, there is need for that among the rank and file of the men who pass the claims, not merely the heads of the department. It must reach the bulk of the men who do the work.

Another thing, the claim men must be made comfortable. They must not be made to smart for their ignorance in the field. I fully realize that no one would consciously make a man feel uncomfortable, but you must keep in mind the situation existing. You with this body of detailed and specialized knowledge, and the other man without it, may create misunderstanding unless handled with care and tact.

We would like to see you get your medical opinions before us in medical language. There is a tendency to speak to us

in terms of legal liability. I think that is understandable. An intelligent physician cannot be expected to do much of this work and not think through to the ultimate conclusion of whether a claim is good or bad, but it is necessary that we have a clear picture of the medical processes by which you arrive at your legal conclusions. It must be remembered that good medicine is not necessarily good law. Many times it is directly the opposite. We are dealing in the business where, even though you are right, you may very well be wrong. We are dealing in a situation where the final arbiter is not the Claims Department nor the Medical Department, but some court that has jurisdiction over the matter.

Do not object to a little questioning sometimes. Remember that the claim man has to put his name on the case and assume the final responsibility for the action of the company. If you were signing some of these, you might like to have a point of view elucidated a little more fully than it is in some of the memoranda that are received.

Furthermore, there is the possibility that you might be wrong. Perhaps in perusing some two or three files accumulated over the year, some vital factor that is of moment to the determination of the claim has been overlooked and, when brought to your attention, will decidedly affect the decision on the claim.

And above all, I would leave this with you if everything else be forgotten. I think it is highly essential that these discussions be carried on in a spirit of kindness, tolerance, and understanding, and in a manner that will promote patient effort on the part of the men in the claims group. This alone will be productive of the results that we all desire.

Dr. McGanity—Thank you very much, Mr. Heller. I am sure we will take the advice of Mr. Heller to heart, in connection with our claims in the future, and try to make our opinions more intelligent to the medical end of the Claims Department.

The next speaker is Mr. John G. Kelly, Assistant General Counsel of The Mutual Life Insurance Company of New York. He is going to speak to us on the "Relationship of the Medical Director to the Home Office Counsel." I will say, in connection with the questions again, we are going to interrupt at one o'clock for lunch and we hope that you will keep your questions in connection with these problems that are discussed today until the lunch is over, and we can continue immediately after lunch. Mr. Kelly!

MR. JOHN G. KELLY—The primary function of a life insurance company is, of course, payment of the benefits provided for by its contracts. Action on the vast majority of cases presents no particular problem. It consists largely of being assured of certain undisputed facts on which liability depends. I recently obtained figures regarding the 1946 double indemnity experience of a group of representative life insurance companies. Lawsuits seeking payment of the accidental death benefit which had been declined by the company represented only 2.5 per cent of the total amount of cases acted upon. I mention this lest our consideration of controversial claims appear to give an exaggerated impression of the numerical relationship of such cases to the total picture.

However, though they be a small minority, there always will be some cases in which the individual insured or his beneficiary and the insurance company (acting on behalf of all of its policyholders) do not see eye to eye. These are the ones that are of particular interest because of the challenge they present. Dr. Filson has referred to this group of cases, saying:

> "When in any such case, after it has been fully developed there is a decision that merit is not established there must be a synchronous and parallel determination of the means and material through which such a decision can, if necessary, be defended."

It is my purpose to comment on the co-operative effort of the Medical Director and Home Office legal counsel on

which successful development of the "means and material" to defend the denial of liability in such cases depends.

Some years ago, my Company prepared a manual to aid its Inspectors in handling disability claims. In the foreword to this manual we said:

> "It is difficult to obtain judgments from courts or juries on a presentation alone of medical testimony which tends solely to contradict the testimony of the plaintiff's medical witnesses. For the jury or court to find in our favor without something more than the showing of a contradiction in the testimony of medical witnesses requires in effect a holding that the plaintiff's doctors either are testifying falsely, are incompetent, or are mistaken in their diagnosis, and that our medical witnesses alone are to be believed or have correctly diagnosed the case. Courts and juries will seldom adopt such a view unless supported by convincing evidence. Whenever possible we must therefore supplement our presentation of medical testimony in three important ways:—first, by breaking down the testimony of the plaintiff's medical witnesses through studied cross-examination directed to disclosing inaccuracies, contradictions or other weaknesses in their testimony; second, by presenting to the court written admissions over the signature of the insured from which an inference contrary to the claim of total disability may reasonably be drawn; third, by presenting testimony of lay witnesses having knowledge of his activities."

This quotation aptly emphasizes the basic assumption that I think must be made—that the successful defense of a medical legal issue demands something more than the mere presentation of medical testimony on behalf of the insurance company which contradicts that presented on behalf of the insured. The quotation suggests three methods by which the Company's medical testimony should be supplemented. We are, of course, only concerned with the first of these:

> "By breaking down the testimony of the plaintiff's medical witnesses through studied cross-examination directed to disclosing inaccuracies, contradictions, or other weaknesses in their testimony."

The method by which this may be accomplished and the

part that the Medical Director plays in aiding in this accomplishment, will, perhaps, best be illustrated by a specific example. Let us assume a case of a claim for total disability. After payment of disability benefits for a period of years the company has denied further liability. The company has no knowledge of any gainful or other significant activity on the part of the insured. The sole issue is a conflict of medical opinion, with the insured being supported by three physicians and the company relying on the opinions of its medical experts. By what method may the "means and material" of successful defense be best developed? I recognize that differences in the organizational set-up of various companies will affect the extent to which the handling I suggest may be feasible. I am assuming an organizational set-up such as exists in my own company in which Home Office attorneys personally handle the trial of litigation in our immediate vicinity and to the extent that it is practicable pass on to local counsel throughout the country the methods and technique worked out here in actual practice.

It has seemed to us that the initial step is for Home Office counsel to obtain a working knowledge of the medical questions involved. Some lawyers may have a tendency to regard medical questions as entirely out of their field, being expressed in technical terms not readily understandable by an attorney. It has been our experience, however, that an attorney, with proper assistance and guidance from his Home Office medical associates, may attain a reasonable degree of proficiency in understanding—and, in fact, even in talking —the technical language of medicine. Diligent use of a good medical dictionary should enable the attorney to translate all of the medical terms in the file before him into words readily understandable by him. Such a working knowledge of the terminology is necessary not only as a prerequisite to any real analysis of the questions involved, but is the only means by which counsel can hope to develop the necessary understanding on the part of a court and jury.

Having obtained a working knowledge of the medical terms, the attorney is prepared to read with considerable comprehension relevant medical literature. With the assistance of a co-operative Medical Director who will direct his attention to pertinent articles on the particular medical problems involved, the attorney will not only develop a greater understanding of the precise question, but is in a position to avail himself of references which prove most useful on cross-examination of the plaintiff's medical witnesses. Extracts from medical texts and magazines may be brought to the attention of a witness to show that there is well-founded medical opinion which contradicts views he has expressed. Occasionally, you will find that a prospective medical witness for the insured has himself written either in a medical text or in an article in a medical magazine along lines which are inconsistent with the position he is taking in the particular case. Cross-examination on the basis of such prior writings of the prospective medical witness are, of course, of inestimable value.

In addition to familiarizing himself with pertinent medical literature on the questions involved, the attorney should also check the standing of the insured's prospective medical witnesses through medical directories. One of the considerations which may incline a jury to accept the testimony of one medical witness in preference to that of another may be the fact that one physician has obvious qualifications in a particular specialty which are not shared by another.

A study of medical texts and articles will enable counsel to acquaint himself with the criteria for the establishment of a sufficient degree of impairment from a particular disease to constitute total disability. It will enable him to distinguish a normal finding from an abnormal one. An attorney prepared to demonstrate the extent to which the findings of the insured's physicians are normal is, of course, in a position to make a real attack upon the opinions of plaintiff's medical witnesses. If it is clear that they are really offering nothing more than conclusions which are not supported by specific

findings on their own examinations, the value of their opinions is materially weakened and the jury is more likely to accept the logic of the company's medical testimony. In this connection the proofs of claim which an insured submits from time to time are most helpful and deserving of careful study. Familiarity with the normal will enable counsel to emphasize physical findings which have repeatedly been reported as within normal limits and results of special examinations which, likewise, have been negative.

Over and above the extent to which a working knowledge of the medical problems involved facilitates the attorney's handling of the case in the manner already indicated, it has been my own experience that it serves as a very definite check on any medical witness who might otherwise be inclined to go to extremes in his testimony, although unsupported by real medical facts. If it is quite clear that counsel trying the case is unaware of the meaning of medical terms, some witnesses will take advantage of the opportunity to express opinions which they would with some hesitancy be willing to present to a committee of their fellow physicians. On the other hand, clear indication that counsel has an adequate understanding of what the medical witness is talking about, and demonstrates that understanding by intelligent and planned cross-examination, has a very definite limiting effect on any tendency on the part of the medical witness to carry his testimony beyond the point supportable by the findings in the case.

All of this may appear to emphasize the work of the lawyer rather than something to which the Medical Director may make a real contribution. However, it has been our experience that, while the spade-work must be done by the attorney, the assistance and guidance of the Medical Director are essential. I have already mentioned the aid the Medical Director may furnish in calling the attention of the attorney to sources of medical literature which will be helpful. In addition, the Medical Director can aid in the interpretation of the medical

facts in the company's file which the attorney is seeking to understand. He can answer the numerous questions which are bound to arise in the mind of the attorney in the course of this attempt to achieve a real understanding of the medical data involved. He can point out the ways in which the opinions expressed by the plaintiff's physicians are unsupported by the findings they have made. Finally, and most important, the Medical Director can and should review the cross-examination that counsel has prepared with a view to assuring its medical accuracy and enabling counsel to avoid any pitfalls into which his own limited knowledge of the medical questions involved might otherwise lead him.

The approach suggested above is based on a controverted disability claim. The method applies to a great extent to the handling of a controverted double indemnity claim. Here, again, a real familiarity with the medical terminology is requisite. Careful consideration of the medical data submitted on behalf of the beneficiary may point to a real weakness in the conclusions drawn or an inconsistency between the specific findings and the opinion that has been rendered. Here, again, medical literature, medical texts and magazines are most helpful in demonstrating the unsoundness of a particular conclusion.

Perhaps nothing that I have said today is news to any of you. However, occasionally I have talked to attorneys who regard a lawsuit which presents solely a medical question as hopeless from the standpoint of the company. It has been our experience, however, that such need not be the case, if the approach suggested above is diligently followed.

DR. MCGANITY—Thank you, Mr. Kelly. It is always a great privilege to hear from the legal end of the profession. I envy them, at times, their ability to do just what Mr. Kelly has talked about, and that is to take a set of textbooks and spend an evening with it, with the appropriate stimulation, and be able to get up the next morning and, word for word,

quote from the textbook. It is something for which we of the medical profession can admire them. It is probably part of their training, part of their work, but it is certainly an attribute that I have always envied the legal profession. Thank you again, Mr. Kelly.

PRESIDENT JIMENIS—Please come to order, gentlemen. Is Dr. Reynold C. Voss, Chairman of the Medical Section of the American Life Convention, present? We were hoping that he would find a moment to address us. I understand that he was here and of course we were delighted to have him with us, but he may come in later and if he does we will certainly have him speak to us. We will proceed now with the forum which began this morning. Dr. McGanity!

DR. MCGANITY—Ladies and gentlemen, we have had the privilege of hearing from three of the speakers on our forum at this session. We have saved the *pièce de résistance* for this afternoon. I know that you will all be glad to hear from Dr. H. Maynard Rees who is Research Director of Medical Services Insurance of the John Hancock Mutual Life Insurance Company. He is going to talk to us on "Claims in Group Casualty Coverages" which, I think, is very interesting to most of us, particularly those concerned with casualty insurance. I know that this is a most interesting subject to us in Canada. We have had a committee working on this problem for some time in an effort to smooth out the criticisms and the difficulties which arise in obtaining correct and sufficiently accurate reports in connection with out disability claims and our casualty work. I suspect from what Dr. Rees has told me that he is going to talk on that particular subject. I am sure it will be interesting to all of us.

I take great pleasure in introducing to you Dr. H. Maynard Rees of the John Hancock Mutual Life Insurance Company. Dr. Rees!

DR. H. MAYNARD REES—My treatment of this subject will be confined to a review of insurance company activities in

co-operating with organized medicine during the past year or two, as they affect group insurance claim procedure.

In the fall of 1946 representatives of seven insurance trade associations met with the American Medical Association in Chicago to determine what areas there were in which the insurance companies and the doctors could co-operate in order to improve medical and surgical care insurance. Out of this meeting there developed a permanent committee of the insurance associations known as the Conference Committee on Health Insurance. This committee is also interested in hospitalization, although as yet contact has not been established with the American Hospital Association.

One of the requests made by the Council on Medical Service of the A. M. A. at the joint meeting was that claim procedure be simplified so that it would be less of a burden on the practicing physician. A subcommittee of the Conference Committee was appointed to work on the subject and this subcommittee was joined by representatives from this association and also from the Association of Life Insurance Counsel and the International Claim Association. This provided the services of specialists in all the aspects of claims: legal, medical and administrative.

The subcommittee worked all last winter and produced model physicians' statements to be used in surgical and weekly indemnity claims. The forms sponsored by the subcommittee have been approved by the parent body—the Conference Committee on Health Insurance—and are destined to be presented to the A. M. A. for approval at an early date.

The method pursued in studying and drawing up these model physicians' statements was to obtain from all companies writing any substantial amount of group insurance their claim forms, and to analyze these and compare the questions asked by each company in each form of coverage. All questions which appeared on these claim forms were discussed and none

was eliminated without what appeared to the committee to be good reason.

In drawing up the resulting forms, the committee had certain objectives in mind, the first of which was clarity. Each question should be so phrased that there would be no doubt about the information which it was desired to elicit. Secondly, questions should be so phrased as not to be offensive to the medical profession, nor to reflect a suspicious attitude which would seem to question the integrity of the doctor. Finally, with one exception which I will comment on, each question should be one which the doctor could properly answer from the information which his professional relations with the patient would make available to him. The exception relates to the establishing of the period of disability. Since a positive statement of fact is hardly possible on this subject, the question was so phrased as to ask the doctor to express an opinion only.

The committee believes that the forms are as simple as they can be made at this time and still satisfy a large majority of the companies. They are a sort of least common denominator. Since the policies and claim procedures of the larger companies vary considerably, it is inevitable that there should be some cases in which the forms are not as simple as those now in use by one or two of these companies. It is felt, however, that the improvement as far as the doctor is concerned which will be brought about by uniformity in the order and form of these questions is more important than the elimination of a line or two from the form. Force of habit is of great importance in the filling out of forms and standardization should save the doctor both time and mental effort.

In submitting these forms to the A. M. A., the Conference Committee feels that it is important that they be considered only as an evolutionary step in the improvement of medical reporting. The insurance companies would like to have them approved, and that approval duly publicized so that the whole

profession will have knowledge of it. It will be emphasized, however, that the companies do not consider these forms to be the ultimate development, and do not desire a form of approval which might tend to freeze them in their present shape. The committee proposes to continue its studies on claim reporting as the general field of medical care insurance unfolds.

It should be borne in mind that neither the Conference Committee nor its subcommittee has any power to bind companies or associations of companies to the use of these or any other forms. No commitments have been made on behalf of any of the companies that took part in this study and none have been sought. If these forms are approved by the A. M. A., their adoption by individual companies will be on a purely voluntary basis.

At this point it is fitting to mention the accomplishments of the companies writing personal accident and health policies in this same field. The Health and Accident Underwriters Conference, working with the International Claim Association, has produced a hospital report form and two forms for the attending physician's statement: one for use in sickness and one for accidents. The hospital report form has been approved by the A. H. A. and the physician's forms by the A. M. A. These forms are now in extensive use among companies writing this type of insurance.

Another field which the Conference Committee on Health Insurance sought to develop with the A. M. A. was that of a better surgical fee schedule. It was hoped that the doctors might take the initiative in developing a fee schedule in which the relative value only of surgical operations was determined. Such a schedule would be in points or percentages and would not set any dollar values. At first it appeared that the A. M. A. was very receptive to this suggestion, but it soon became evident there were too many conflicting interests within the doctors' own organizations to make such a piece of work feasible. As the matter now stands, the Council on Medical

Service has let it be known that no model fee schedule can be expected from them.

Before reaching this decision, however, they had asked the insurance companies to undertake a study of their own experience with surgical claims. A number of companies writing a large volume of group insurance are now in process of putting their experience into such shape that it will serve as the basis for a study by the Actuarial Society of America. This study is to be directed toward developing three sets of data: first, the relative frequency of various surgical procedures in insurance company experience. It is important to distinguish between the relative frequency of an operation where non-occupational accidents and illness only are considered, as compared with its frequency in general surgical or hospital practice. Such surgical data as are now available to the profession are naturally of this latter type. Second, the average surgeon's charge for various procedures. It is to be hoped that in developing this information, the Society will do so by geographical regions, since very considerable differences exist between sections of the country. The third classification is that of the average claim payment which has been made for each procedure.

This study will reveal the most outstanding points where present company schedules are inadequate in meeting a reasonable share of the patient's surgical bill and will make it possible to revise fee schedules accordingly. It is too early to say whether insurance companies will do this as a joint undertaking; but there is no question but that many advantages would flow from such a uniform schedule.

So far we have been concerned with developments at the national level, which seem inevitably to be of slow evolution. At the level of the state medical societies, developments have been more rapid. The State Society of Wisconsin put into effect a plan of surgical care insurance underwritten by private insurance companies almost two years ago. In so doing it

proceeded with the help of certain local insurance companies only. In the spring of this year, the Rhode Island Society appealed directly to the Conference Committee on Health Insurance for help in developing such a plan. The result was that a subcommittee representing all of the various types of insurance carriers met repeatedly with the doctors and helped them draw up a set of Principles to be promulgated by the Society for the guidance of insurance companies in preparing policies which would be acceptable to the Medical Society. In creating such a plan, it was important to avoid some of the pitfalls into which the pioneer Wisconsin Plan had fallen. Among these was clear violation of the federal Anti-Trust Law as regards the development of uniform premium rates; the setting up of certain rigidities of coverage both in surgical and hospitalization expense insurance which hampered the participating companies in their efforts to sell; and, finally, a cumbersome system of joint adjudication of claims whenever a difference of opinion arose between either the insurance company and the doctor, or the doctor and his patient. The Rhode Island Plan has been adopted by the House of Delegates in that state and the Wisconsin House of Delegates has recently taken action revising its Plan along virtually identical lines. It is expected that several other state societies will follow suit in the near future.

The only provision in the Rhode Island type of plan which affects claims is that which gives the medical society the right to review any payment made by an insurance company for an unlisted procedure. Since the society has drawn up a reference fee schedule much longer and more detailed than that which is expected to be published as a part of the insurance policy, these cases should not be too numerous even at the start. It is further provided that where the society determines that the fee paid by the insurance company is incorrect for such an unlisted procedure, it may proceed to establish a fee for the operation in question: (a) this fee shall be consistent with the general level of fees in the master schedule;

and (b) this fee, once determined, shall then become a part of the master schedule. This will prevent its becoming a perennial question subject to redetermination each time it comes up.

The orientation of medical society-sponsored plans in general, however, will have another effect on fee schedules which will be reflected in claims. In order to meet the competition of local service plans sponsored by Blue Cross and Blue Shield organizations, the doctors in Wisconsin and Rhode Island have found it advisable to agree with their own society that they will accept the fees payable by the insurance companies under the master schedule of benefits as full compensation for their services in the case of individuals whose income falls within a predetermined bracket. When the question of full compensation is raised so that the insurance company's payment is no longer a pure indemnity payable to the insured as a contribution toward his doctor's bill, a medical society at once begins to question the adequacy of the level of the fee schedule. Inflation is not a phenomenon which can be confined to the price of goods alone; it affects also the doctor's evaluation of his services. Even though the full payment provision is designed to apply only to the group of the medically indigent who otherwise probably would be unable to pay much of anything for their surgical care, still the doctor fears the influence of any such schedule on the general evaluation which the public will place upon his services. There is, moreover, the unfortunate element of bitter rivalry with the so-called Service Plans, and their fee schedules. We may, therefore, expect pressure for general increases in surgical fees as the result of these state society plans.

At a still lower level, local associations of doctors have sporadically made demands which affect claim procedure. The most common of such demands has had to do with a fee to be paid to the doctor for filling out routine papers in connection with group claims. Some hospitals have tried to put a similar charge into effect.

It has so far been the attitude of most group-writing companies that the furnishing of proof of claim is the duty of the insured and that there is no provision in their contracts for the payment of a fee to either doctor or hospital for this service. This does not, of course, apply where special examination of the insured is requested by the company. It has so far been possible to confer with such local societies and to convince them of the propriety of the insurance company's stand. There are usually other grievances associated with the demand for a fee which can be compromised to the doctor's satisfaction. A common one is dissatisfaction with the multiplicity and complexity of claim forms; this should largely be eliminated in future by A. M. A. approval of model forms. Another complaint has to do with excessive investigation by representatives of the insurance company, which takes up an undue amount of the busy doctor's time. In dealing with such a situation it has been found valuable to arrange for a doctors' committee with periodically changing personnel which will consider methods of improving claim procedure from the doctor's standpoint, investigate cases of abuse by certain doctors of their responsibility in certifying to the existence or termination of disability, and appoint impartial examiners. Where such a committee is composed of the better elements of the local profession, their policing of their fellow practitioners is much more effective than anything that can be done by the insurance carrier.

DR. MCGANITY—Thank you very much, Dr. Rees.

We have some time now, as our next speaker is not available at the moment. I am sure that this last paper is bound to create, in the minds of some, questions that you will want to ask.

The only question I have here at the present time is one addressed to Mr. Heller by Dr. James R. Gudger, asking: "Can you state which source of information is considered most valuable in average medical cases, that from the company

investigator, the company examiner, the consultant, or the attending physician?"

MR. HELLER—I do not think that will permit any single answer. I think that the answer changes with the nature of the case. There are some cases, for instance, of a serious condition with largely subjective signs of the illness which we are unable to verify on examination, but everything in the case indicates that we are dealing with an insured and a doctor of highest repute. In such a case as that, it might well be that the attending physician's statement would be determinative of the matter, at least for a while. Take that very case and find a situation where the medical testimony from the attending physician and our examiner is the same, but an investigator reveals an actual state of affairs showing physical activity on the part of the insured wholly out of line with the medical evidence presented. In that case, the investigator's viewpoint would perhaps be the most important. In some of these cases that are rather carelessly prepared by attending physicians, where the investigator's report is of no great moment, the report of the company examiner or the specialist called in by the company might well be the determining factor.

DR. MCGANITY—Thank you very much, Mr. Heller.

Is there any other question in regard to these four papers? I judge, then, that you are all satisfied with the projected scheme that Dr. Rees has outlined to you for the simplification of reporting and keeping records of the casualty business in your various companies.

If there are no further questions, then I would like at this time to express my personal thanks to all the contributors to this forum today. As I said at the beginning, the arranging of this program was entirely the effort of your executives, and I have had the honor of presiding at what I think has been one of the best forums that we have had in connection with this Association since I have been a member. I hope the papers

have met with your approval, and I am sure we could not have had four better speakers.

That concludes our forum, and I am going to ask our President, Dr. Jimenis, to come forward and take over the chair again and conclude the meeting.

PRESIDENT JIMENIS—Our thanks are due Dr. McGanity for the able and gracious manner in which he has conducted this forum. I enjoyed it myself, and I am sure you all did, too.

Gentlemen, our meeting is drawing to a close, and I think we are all grateful to the clinicians and guests who have helped us along and brought their own knowledge and given us such interesting papers.

For my own part, I am deeply appreciative of the help that has been afforded to me by our officers, principally our secretary, Dr. Henry B. Kirkland, and everyone else who has had any part in this meeting.

It remains now only to call to the chair our President-elect, Dr. Albert J. Robinson, of the Connecticut General Life Insurance Company. Dr. Robinson!

DR. ALBERT J. ROBINSON—Dr. Jimenis, I know that I express the unanimous sentiment of this meeting when I say that you are to be congratulated on the excellence of the program and the meeting in its entirety, and may I suggest to the members that we so express ourselves in a rising vote of thanks to Dr. Jimenis.

As for myself, I deeply appreciate the confidence that you have imposed in me in voting me as your President for the next year. I shall strive to have a program which I hope will measure up to the one just concluded. I shall also attempt to continue the splendid traditions of the Medical Directors Association because this association plays such an important part in the overall conduct of the great institution of life insurance.

Is there further business to come before this meeting? If not, I shall entertain a motion to adjourn.

* * *

A motion to adjourn was made, seconded and carried, and the meeting was adjourned.

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The following members and guests were present at some time during the sessions: Doctors C. B. Ahlefeld, H. H. Amiral, K. W. Anderson, T. D. Archibald, T. M. Armstrong, W. B. Aten, D. R. Auten, J. A. Avrack, H. A. Bancel, G. H. Barber, N. J. Barker, C. M. Barrett, J. R. Beard, E. W. Beckwith, J. E. Bee, M. B. Bender, R. A. Benson, C. C. Berwick, J. R. Biggs, W. R. Bishop, N. R. Blatherwick, J. E. Boland, William Bolt, E. C. Bonnett, J. T. Bowman, W. M. Bradshaw, K. F. Brandon, A. W. Bromer, Leslie Brown, E. R. Bush, B. F. Byrd, J. T. Cabaniss, E. J. Campbell, P. E. Carlisle, D. W. Carter, V. S. Caviness, J. P. Chapman, P. H. Charlton, H. E. Christensen, E. E. Clovis, A. D. Cloyd, H. A. Cochran, Jr., N. B. Cole, B. L. Coley, H. L. Colombo, B. R. Comeau, T. E. Cone, Jr., F. R. Congdon, J. L. Cook, P. M. Cort, H. I. Crooker, R. M. Daley, H. D. Delamere, P. G. Denker, E. J. Dewees, E. G. Dewis, T. H. Dickson, H. W. Dingman, A. H. Domm, J. P. Donelan, H. R. Dove, W. W. Dow, L. I. Dublin, T. C. Dunlop, T. M. Ebers, L. B. Ellis, A. H. Faber, J. G. Falconer, R. K. Farnham, H. H. Fellows, W. E. Ferguson, R. M. Filson, R. W. Finegan, V. J. Fingar, P. M. L. Forsberg, E. M. Freeland, D. G. Friend, H. M. Frost, F. I. Ganot, I. K. Gardner, D. S. Garner, J. T. Geiger, W. M. Genthner, E. E. Getman, J. M. Gilchrist, R. A. Goodell, H. W. Goos, A. S. Graham, R. J. Graves, R. S. Gubner, J. R. Gudger, Llewellyn Hall, F. T. Hallam, G. A. Harlow, Frank Harnden, L. E. Hathaway, Jr., W. C. Hausheer, H. M. Hawkins, J. K. P. Hawks, W. D. Heaton, Milton Helpern, O. C. Hendrix, E. V. Higgins, W. L. Hilliard, E. C. Hillman,

Jr., I. E. Hoffman, J. C. Horan, J. H. Humphries, B. L. Huntington, J. J. Hutchinson, J. R. B. Hutchinson, W. G. Hyde, A. S. Irving, J. G. Irving, W. A. Jaquith, A. O. Jimenis, H. R. John, H. J. Johnson, J. W. Johnson, Jr., H. B. Jones, E. P. Joslin, E. A. Keenleyside, N. R. Kelley, E. F. Kerby, C. E. Kiessling, D. G. Kilgore, H. B. Kirkland, F. H. Lahey, Phillips Lambkin, P. H. Langner, Jr., L. G. LaPointe, I. C. Lawler, L. H. Lee, E. P. Leeper, H. R. Leffingwell, E. H. Lindstrom, J. M. Livingston, H. C. McAlister, F. A. McChesney, George McCreight, C. B. McCulloch, F. J. McGurl, W. G. McLaughry, George McLean, L. L. McLellan, R. E. McLochlin, W. J. McNamara, Charles Maertz, H. R. Magee, S. J. N. Magwood, R. W. Mann, E. B. Milam, J. T. Montgomery, J. F. Moore, Jr., S. A. Narins, R. A. Nelson, E. C. Noble, Eduard Novak, M. J. O'Brien, Herbert Old, M. I. Olsen, B. H. Olson, I. H. Page, W. C. Page, C. B. Parker, A. E. Parks, J. E. Patterson, D. S. Pepper, C. A. Peters, Cullen Pitt, R. W. Pratt, M. A. Puzak, P. K. Rand, O. S. Randall, J. H. Ready, Rezin Reagan, C. L. Reeder, H. M. Rees, P. V. Reinartz, David Reisner, W. A. Reiter, W. M. Reynolds, F. X. Riley, G. P. Robb, D. C. Roberts, Tom Robertson, A. J. Robinson, H. B. Rollins, F. W. Rolph, R. C. Roskelley, Gordon Ross, R. L. Rowley, W. W. Rucks, Edward Ruge, J. F. Sadusk, Jr., K. F. Schaefer, B. T. D. Schwarz, B. E. Schwarz, R. C. Secor, D. L. Selby, T. S. Sexton, Hall Shannon, J. T. Sheridan, D. M. Shewbrooks, R. R. Simmons, J. M. Smith, W. A. Smith, F. L. Springer, H. F. Starr, J. B. Steele, D. F. R. Steuart, L. Q. Stewart, I. R. Stidger, S. J. Streight, N. A. Sullo, L. G. Sykes, B. C. Syverson, K. J. Thomson, W. E. Thornton, Joseph Travenick, Jr., Wallace Troup, Maurice Turcotte, H. B. Turner, H. E. Ungerleider, B. W. Vale, R. C. Voss, Murray Wagman, F. A. Waldron, C. F. Warren, R. L. Weaver, Jefferson Weed, D. E. W. Wenstrand, S. S. Werth, O. B. Wight, E. S. Williams, R. L. Willis, A. A. Wills, Jr., A. C. Wilson, M. C. Wilson, G. E. Woodford, L. S. Ylvisaker, A. W. Young, R. W. Zinkann, and Messrs. E. J. Bohne, Edwin Dowling, E. J.

Hardin, R. T. Heller, J. C. Higdon, Arthur Hunter, W. N. Hutchinson, L. J. Kalmbach, J. G. Kelly, Edward King, G. C. Kingsley, Jr., H. J. Laramee, J. A. McLain, H. H. Marks, A. P. Morton, Charles Northrup, E. E. Rhodes, Pearce Shepherd, O. G. Sherman, F. W. White, J. C. Wilberding, and Miss A. M. Lyle.

Total attendance at all sessions, 265.

In Memoriam

Deceased Since Fifty-fifth Annual Meeting

Donald B. Cragin, M. D.
Aetna Life Insurance Company
Died July 13, 1947.

Hugh W. Crawford, M. D.
John Hancock Mutual Life Insurance Company
Died September 25, 1947.

William L. Mann, M. D.
Great-West Life Assurance Company
Died May 31, 1947.

Edward L. Mathias, M. D.
Kansas City Life Insurance Company
Died February 3, 1947.

Joseph O. Segura, M. D.
Lamar Life Insurance Company
Died October 26, 1946.

Archibald R. Stone, M. D.
Midland Mutual Life Insurance Company
Died April 13, 1947.

DECEASED MEMBERS

John L. Adams, M. D.	New York, N. Y.
Charles D. Alton, M. D.	Hartford, Conn.
Malcolm O. Austin, M. D.	San Francisco, Calif.
Walter C. Bailey, M. D.	Boston, Mass.
Henry A. Baker, M. D.	Kansas City, Mo.
A. W. Barrows, M. D.	Hartford, Conn.
John T. J. Battle, M. D.	Greensboro, N. C.
Wesley W. Beckett, M. D.	Los Angeles, Calif.
Charles D. Bennett, M. D.	Newark, N. J.
Emmanuel P. Benoit, M. D.	Montreal, Can.
Charles Bernacki, M. D.	New York, N. Y.
Thomas W. Bickerton, M. D.	New York, N. Y.
Albert W. Billing, M. D.	New York, N. Y.
Wilton F. Blackford, M. D.	Louisville, Ky.
David N. Blakely, M. D.	Boston, Mass.
Robert J. Blanchard, M. D.	Winnipeg, Man.
Harold E. Bogart, M. D.	New York, N. Y.
Frederick G. Brathwaite, M. D.	New York, N. Y.
William R. Bross, M. D.	New York, N. Y.
Chauncey R. Burr, M. D.	New York, N. Y.
Robert L. Burrage, M. D.	Newark, N. J.
James Campbell, M. D.	Hartford, Conn.
Willard B. Carpenter, M. D.	Columbus, Ohio
Frank W. Chapin, M. D.	New York, N. Y.
Frederick W. Chapin, M. D.	Springfield, Mass.
Ferdinand E. Chatard, M. D.	Baltimore, Md.
Charles L. Christiernin, M. D.	New York, N. Y.
Henry Colt, M. D.	Pittsfield, Mass.
Henry W. Cook, M. D.	Minneapolis, Minn.
Donald B. Cragin, M. D.	Hartford, Conn.
Thomas C. Craig, M. D.	New York, N. Y.
Hugh W. Crawford, M. D.	Boston, Mass.
Hamilton C. Cruikshank, M. D.	Toronto, Canada
Edward Curtis, M. D.	New York, N. Y.

DECEASED MEMBERS

Clark W. Davis, M. D.	Cincinnati, Ohio
William B. Davis, M. D.	Cincinnati, Ohio
Charles A. Devendorf, M. D.	Detroit, Mich.
Henry K. Dillard, M. D.	Philadelphia, Pa.
Frank Donaldson, M. D.	Baltimore, Md.
Percy G. Drake, M. D.	Hartford, Conn.
Edwin W. Dwight, M. D.	Boston, Mass.
James B. Eagleson, M. D.	Seattle, Wash.
Z. Taylor Emery, M. D.	New York, N. Y.
Joseph E. Engelson, M. D.	New York, N. Y.
Francis C. Evers, M. D.	New York, N. Y.
William G. Exton, M. D.	Newark, N. J.
Robert H. Feldt, M. D.	Milwaukee, Wis.
John W. Fisher, M. D.	Milwaukee, Wis.
Paul FitzGerald, M. D.	Newark, N. J.
Thomas A. Foster, M. D.	Portland, Me.
Robert A. Fraser, M. D.	New York, N. Y.
Samuel W. Gadd, M. D.	Philadelphia, Pa.
Homer Gage, M. D.	Worcester, Mass.
Thomas H. Gage, M. D.	Worcester, Mass.
Donald M. Gedge, M. D.	New York, N. Y.
Walter R. Gillette, M. D.	New York, N. Y.
Frank S. Grant, M. D.	New York, N. Y.
Frederick L. Grasett, M. D.	Toronto, Can.
Landon Carter Gray, M. D.	New York, N. Y.
Frederick W. Hagney, M. D.	Newark, N. J.
Ignatius Haines, M. D.	Boston, Mass.
George C. Hall, M. D.	Richmond, Va.
Joseph B. Hall, M. D.	Hartford, Conn.
Edward H. Hamill, M. D.	Newark, N. J.
William W. Hitchcock, M. D.	Los Angeles, Calif.
Angier B. Hobbs, M. D.	New York, N. Y.
William W. Hobson, M. D.	Pittsburgh, Pa.
Donald C. Hoffman, M. D.	New York, N. Y.

DECEASED MEMBERS

Edgar Holden, M. D.	Newark, N. J.
John Homans, M. D.	Boston, Mass.
John Homans, 2d, M. D.	Boston, Mass.
J. Charles Humphreys, M. D.	Philadelphia, Pa.
Abel Huntington, M. D.	New York, N. Y.
Ross Huston, M. D.	Des Moines, Iowa
Henry H. Hutchison, M. D.	Toronto, Can.
Lefferts Hutton, M. D.	New York, N. Y.
Charles E. Iliff, M. D.	Cincinnati, Ohio
Phineas H. Ingalls, M. D.	Hartford, Conn.
Charles B. Irwin, M. D.	Chicago, Ill.
Arthur Jukes Johnson, M. D.	Toronto, Can.
William M. Jones, M. D.	Greensboro, N. C.
John M. Keating, M. D.	Philadelphia, Pa.
Edward B. Kellogg, M. D.	Boston, Mass.
Frank W. Kenney, M. D.	Denver, Colo.
William W. Knight, M. D.	Hartford, Conn.
Edward Lambert, M. D.	New York, N. Y.
John B. Lewis, M. D.	Hartford, Conn.
Ernest H. Lines, M. D.	New York, N. Y.
John M. Little, M. D.	Boston, Mass.
Robert L. Lounsberry, M. D.	Binghamton, N. Y.
Carl Lovelace, M. D.	Waco, Texas
Henry P. Lyster, M. D.	Detroit, Mich.
Lewis F. MacKenzie, M. D.	Newark, N. J.
Milton T. McCarty, M. D.	Frankfort, Ind.
Charles N. McCloud, M. D.	St. Paul, Minn.
Francis A. McGreen, M. D.	New York, N. Y.
Lewis McKnight, M. D.	Milwaukee, Wis.
Thomas H. McMahon, M. D.	Toronto, Ont.
Walter T. McNaughton, M. D.	Milwaukee, Wis.
William L. Mann, M. D.	Winnipeg, Canada
Elias J. Marsh, M. D.	Paterson, N. J.
Henry A. Martelle, M. D.	Hartford, Conn.

DECEASED MEMBERS

Edward L. Mathias, M. D.	Kansas City, Mo.
Allison Maxwell, M. D.	Indianapolis, Ind.
Archibald Mercer, M. D.	Newark, N. J.
Francis D. Merchant, M. D.	New York, N. Y.
William R. Miller, M. D.	Hartford, Conn.
William D. Morgan, M. D.	Hartford, Conn.
John P. Munn, M. D.	New York, N. Y.
William Natress, M. D.	Toronto, Ont.
Charles T. Necker, M. D.	Waterloo, Can.
Edwin M. Northcott, M. D.	Portland, Me.
Ralph B. Ober, M. D.	Springfield, Mass.
Brace W. Paddock, M. D.	Pittsfield, Mass.
Frank K. Paddock, M. D.	Pittsfield, Mass.
J. Allen Patton, M. D.	Newark, N. J.
William O. Pauli, M. D.	Cincinnati, Ohio
William A. Peterson, M. D.	Chicago, Ill.
Joseph E. Pollard, M. D.	Newark, N. J.
William E. Porter, M. D.	New York, N. Y.
Albert T. Post, M. D.	New York, N. Y.
James T. Priestly, M. D.	Des Moines, Iowa
William W. Quinlan, M. D.	Chicago, Ill.
Oliver P. Rex, M. D.	Philadelphia, Pa.
James A. Roberts, M. D.	Toronto, Ont.
Thomas H. Rockwell, M. D.	New York, N. Y.
Oscar H. Rogers, M. D.	New York, N. Y.
Edward K. Root, M. D.	Hartford, Conn.
James F. W. Ross, M. D.	Toronto, Ont.
Charles L. Rudasill, M. D.	Richmond, Va.
Gurdon W. Russell, M. D.	Hartford, Conn.
Robert Sanderson, M. D.	Boston, Mass.
Joseph O. Segura, M. D.	Jackson, Miss.
George R. Shepherd, M. D.	Hartford, Conn.
Arthur L. Sherrill, M. D.	New York, N. Y.
Donald W. Skeel, M. D.	Los Angeles, Calif.

DECEASED MEMBERS

Dewitt Smith, M. D.	Dallas, Tex.
George S. Stebbins, M. D.	Springfield, Mass.
Archibald R. Stone, M. D.	Columbus, Ohio
George S. Strathy, M. D.	Toronto, Ont.
Melancthon Storrs, M. D.	Hartford, Conn.
Brandreth Symonds, M. D.	New York, N. Y.
H. Cabell Tabb, M. D.	Richmond, Va.
Harold F. Taylor, M. D.	Hartford, Conn.
James Thorburn, M. D.	Toronto, Ont.
James D. Thorburn, M. D.	Toronto, Ont.
William Thorndike, M. D.	Milwaukee, Wis.
Paul E. Tiemann, M. D.	New York, N. Y.
Harry Toulmin, M. D.	Philadelphia, Pa.
Frank L. Truitt, M. D.	Indianapolis, Ind.
Henry Tuck, M. D.	New York, N. Y.
John S. Turner, M. D.	Dallas, Tex.
Joseph P. Turner, M. D.	Greensboro, N. C.
S. Oakley Van der Poel, M. D.	New York, N. Y.
Charles A. VanDervoort, M. D.	Philadelphia, Pa.
A. L. Vanderwater, M. D.	New York, N. Y.
Clinton D. W. VanDyck, M. D.	New York, N. Y.
George A. VanWagenen, M. D.	Newark, N. J.
Aaron C. Ward, M. D.	Newark, N. J.
William Perry Watson, M. D.	Newark, N. J.
Joseph H. Webb, M. D.	Waterloo, Ont.
William E. H. Wehner, M. D.	Philadelphia, Pa.
Faneuil S. Weisse, M. D.	New York, N. Y.
George R. Welch, M. D.	New York, N. Y.
Ernest A. Wells, M. D.	Hartford, Conn.
Frank Wells, M. D.	Boston, Mass.
Franklin C. Wells, M. D.	New York, N. Y.
George W. Wells, M. D.	New York, N. Y.
Charles D. Wheeler, M. D.	Worcester, Mass.
A. H. Whitridge, M. D.	Baltimore, Md.

DECEASED MEMBERS

George Wilkins, M. D.	Montreal, Que.
Thomas H. Willard, M. D.	New York, N. Y.
Charles H. Willits, M. D.	Philadelphia, Pa.
Gordon Wilson, M. D.	Baltimore, Md.
G. S. Winston, M. D.	New York, N. Y.
Harry P. Woley, M. D.	New York, N. Y.
Albert Wood, M. D.	Worcester, Mass.
Green V. Woolen, M. D.	Indianapolis, Ind.
John C. Young, M. D.	Detroit, Mich.
Joseph C. Young, M. D.	Newark, N. J.

LIST OF MEMBERS OF THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS

John W. Abbott, M. D.	Paul Revere, Worcester, Mass.
Charles B. Ahlefeld, M. D.	Business Men's, Kansas City, Mo.
George E. Allen, M. D.	National, Montpelier, Vt.
Hiram H. Amiral, M. D.	State Mutual, Worcester, Mass.
Henry H. Amsden, M. D.	United Life and Accident, Concord, N. H.
Karl W. Anderson, M. D.	Northwestern National, Minneapolis, Minn.
Perry A. Anderson, M. D.	Rockford Life, Rockford, Ill.
Thomas D. Archibald, M. D.	T. Eaton, Toronto, Can.
Thomas M. Armstrong, M. D.	Philadelphia Life, Philadelphia, Pa.
William B. Aten, M. D.	Security Mutual, Binghamton, N. Y.
Donald R. Auten, M. D.	New York Life, New York City
J. Albert Avrack, M. D.	United States Life, New York City
Henry A. Bancel, M. D.	Mutual, New York City
G. Holbrook Barber, M. D.	Manhattan, New York City
Norman J. Barker, M. D.	Connecticut General, Hartford, Conn.
Charles M. Barrett, M. D.	Western and Southern, Cincinnati, Ohio
Daniel S. Baughman, M. D.	Security Life and Accident, Denver, Colo.
Carroll C. Beach, Jr., M. D.	Connecticut General, Hartford, Conn.

J. Randolph Beard, M. D.	Mutual Benefit, Newark, N. J.
Edgar W. Beckwith, M. D.	Equitable Life Assurance, New York City
James E. Bee, M. D.	Kansas City Life, Kansas City, Mo.
Roland A. Behrman, M. D.	John Hancock Mutual, Boston, Mass.
J. V. Bell, M. D.	National Fidelity, Kansas City, Mo.
Maurice B. Bender, M. D.	Guardian, New York City
Roy W. Benton, M. D.	Northwestern Mutual, Milwaukee, Wis.
C. Coleman Berwick, M. D.	Metropolitan, New York City
Francis P. Bicknell, M. D.	State Mutual, Worcester, Mass.
J. Rozier Biggs, M. D.	Peoples, Washington, D. C.
Cecil C. Birchard, M. D.	Sun, Montreal, Can.
B. Cosby Bird, M. D.	Preferred, Montgomery, Ala.
William R. Bishop, M. D.	Jefferson Standard, Greensboro, N. C.
Norman R. Blatherwick, M. D.	Metropolitan, New York City
John E. Boland, M. D.	Country, Chicago, Ill.
William Bolt, M. D.	New York Life, New York City
Earl C. Bonnett, M. D.	Metropolitan, New York City
J. Thornley Bowman, M. D.	London Life, London, Can.
William M. Bradshaw, M. D.	Mutual, New York City
William E. Branch, M. D.	Constitution, Los Angeles, Calif.
Kenneth F. Brandon, M. D.	Aetna, Hartford, Conn.
Albert W. Bromer, M. D.	Metropolitan, New York City

C. Frank Brown, M. D.	Southwestern, Dallas, Texas
Frederick R. Brown, M. D.	New England Mutual, Boston, Mass.
Howard B. Brown, M. D.	Massachusetts Mutual, Springfield, Mass.
Leslie Brown, M. D.	Equitable Life Assurance, New York City
William Brueggemann, M. D.	Union Central, Cincinnati, Ohio
Earl R. Bush, M. D.	Western and Southern, Cincinnati, Ohio
Benjamin F. Byrd, M. D.	National Life & Accident, Nashville, Tenn.
Joseph T. Cabaniss, M. D.	Travelers, Hartford, Conn.
Edward J. Campbell, M. D.	New York Life, New York City
Hugh B. Campbell, M. D.	Phoenix Mutual, Hartford, Conn.
Frank H. Carber, M. D.	Mutual, New York City
Paul E. Carlisle, M. D.	Prudential, Newark, N. J.
David W. Carter, Jr., M. D.	Reserve Loan, Dallas, Texas
Verne S. Caviness, M. D.	Occidental, Raleigh, N. C.
Laurence D. Chapin, M. D.	Massachusetts Mutual, Springfield, Mass.
John P. Chapman, M. D.	Girard, Philadelphia, Pa.
Paul H. Charlton, M. D.	Midland Mutual, Columbus, Ohio
Edmund D. Chesebro, M. D.	Puritan, Providence, R. I.
Harry E. Christensen, M. D.	Union Mutual, Portland, Maine
Milton H. Clifford, M. D.	New England Mutual, Boston, Mass.
Elijah E. Clovis, M. D.	Conservative, Wheeling, W. Va.

Augustus D. Cloyd, M. D.	Woodmen of the World, Omaha, Neb.
Harry A. Cochran, Jr., M. D.	Reliance, Pittsburgh, Pa.
Norman B. Cole, M. D.	Baltimore Life, Baltimore, Md.
Irwin E. Colgin, M. D.	Texas Life, Waco, Texas
Harry L. Colombo, M. D.	National Life, Montpelier, Vt.
Thomas E. Cone, Jr., M. D.	Penn Mutual, Philadelphia, Pa.
Frederick R. Congdon, M. D.	Berkshire, Pittsfield, Mass.
H. F. Connally, Sr., M. D.	Amicable, Waco, Texas
Francis V. Costello, M. D.	Mutual, New York City
Neil L. Criss, M. D.	United Benefit, Omaha, Neb.
Howard K. Crutcher, M. D.	United Fidelity, Dallas, Texas
John P. Davis, M. D.	Security Life & Trust, Winston-Salem, N. C.
Harold D. Delamere, M. D.	Crown, Toronto, Can.
J. Emile Desrochers, M. D.	La Sauvegarde, Montreal, Canada
Ernest J. Dewees, M. D.	Provident Mutual, Philadelphia, Pa.
Earle T. Dewey, M. D.	Metropolitan, New York City
Edwin G. Dewis, M. D.	Prudential, Newark, N. J.
Thomas H. Dickson, M. D.	Minnesota Mutual, St. Paul, Minn.
Edward S. Dillon, M. D.	Penn Mutual, Philadelphia, Pa.
Harold W. Dingman, M. D.	Continental Assurance, Chicago, Ill.
Nathaniel P. Doak, M. D.	Great Southern, Houston, Texas
Albert H. Domm, M. D.	Prudential, Newark, N. J.

James P. Donelan, M. D.	Guarantee Mutual, Omaha, Neb.
Gerald D. Dorman, M. D.	New York Life, New York City
William W. Dow, M. D.	Toronto Mutual, Toronto, Canada
James T. Downs, Jr., M. D.	Fidelity Union, Dallas, Texas
Thomas C. Dunlop, M. D.	Manufacturers, Toronto, Canada
Louis B. Dunn, M. D.	Postal, New York City
John T. Eads, M. D.	Penn Mutual, Philadelphia, Pa.
William W. Eakin, M. D.	Standard, Montreal, Canada
Theodore M. Ebers, M. D.	Connecticut Mutual, Hartford, Conn.
H. Glenn Ebersole, M. D.	Illinois Bankers, Monmouth, Ill.
Laurence B. Ellis, M. D.	Boston Mutual, Boston, Mass.
Jack A. End, M. D.	Northwestern Mutual, Milwaukee, Wis.
John A. Evans, M. D.	Metropolitan, New York City
John L. Evans, M. D.	Farmers & Bankers, Wichita, Kan.
Albert H. Faber, M. D.	New York Life, New York City
J. Gilbert Falconer, M. D.	North American, Toronto, Can.
Raymond K. Farnham, M. D.	Metropolitan, New York City
Haynes H. Fellows, M. D.	Metropolitan, New York City
W. Ewart Ferguson, M. D.	Excelsior, Toronto, Can.
William S. Fewell, M. D.	Liberty, Greenville, S. C.
Ralph M. Filson, M. D.	Travelers, Hartford, Conn.

Rexford W. Finegan, M. D.	Metropolitan, New York City
Victor J. Fingar, M. D.	Equitable Life Assurance, New York City
Frederick Fink, M. D.	Franklin, Springfield, Ill.
Harry E. Flansburg, M. D.	Bankers, Lincoln, Neb.
Philip M. L. Forsberg, M. D.	United Life and Accident, Concord, N. H.
Garth E. Fort, M. D.	National Life & Accident, Nashville, Tenn.
John M. Foster, Jr., M. D.	Capitol, Denver, Colo.
Edward M. Freeland, M. D.	New York Life, New York City
Dale G. Friend, M. D.	John Hancock Mutual, Boston, Mass.
Harold M. Frost, M. D.	New England Mutual, Boston, Mass.
F. Irving Ganot, M. D.	Prudential, Newark, N. J.
I. Kenneth Gardner, M. D.	Reliance, Pittsburgh, Pa.
David S. Garner, M. D.	Shenandoah, Roanoke, Va.
J. H. Geddes, M. D.	Northern, London, Canada
John T. Geiger, M. D.	Metropolitan, New York City
William M. Genthner, M. D.	Continental American, Wilmington, Del.
Edson E. Getman, M. D.	New York Life, New York City
Henry W. Gibbons, M. D.	California-Western States, Sacramento, Calif.
John M. Gilchrist, M. D.	Monarch, Springfield, Mass.
Ralph T. Gilchrist, M. D.	Northwestern Mutual, Milwaukee, Wis.
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Manufacturers Life Ins. Co., Toronto, Canada.	T. C. Dunlop, M. D. H. M. Gray, M. D. R. C. Montgomery, M. D.

Maritime Life Assurance Co., Halifax, N. S., Canada	J. G. MacDougall, M. D.
Maryland Life Insurance Co., Baltimore, Md.	G. C. Lockard, M. D.
Massachusetts Mutual Life Insurance Co., Springfield, Mass.	H. B. Brown, M. D. L. D. Chapin, M. D. L. E. Haentzschel, M. D. Gordon Ross, M. D. T. S. Sexton, M. D. Morton Snow, M. D.
Metropolitan Life Insurance Co., New York City	C. C. Berwick, M. D. N. R. Blatherwick, M. D. E. C. Bonnett, M. D. A. W. Bromer, M. D. E. T. Dewey, M. D. J. A. Evans, M. D. R. K. Farnham, M. D. H. H. Fellows, M. D. R. W. Finegan, M. D. J. T. Geiger, M. D. J. C. Horan, M. D. A. O. Jimenis, M. D. H. B. Kidd, M. D. A. J. Lanza, M. D. G. P. Robb, M. D. K. J. Thomson, M. D. Wallace Troup, M. D. E. W. Wilson, M. D.
Michigan Life Insurance Co., Detroit, Mich.	R. W. Scott, M. D.
Midland Mutual Life Insurance Co., Columbus, Ohio	P. H. Charlton, M. D.
Midland National Life Insurance Co., Watertown, S. D.	O. S. Randall, M. D.
Midwest Life Insurance Co., Lincoln, Neb.	E. W. Rowe, M. D.

Minnesota Mutual Life Insurance Co., St. Paul, Minn.	T. H. Dickson, M. D. A. E. Venables, M. D.
Missouri Insurance Co., St. Louis, Mo.	C. D. Magee, M. D.
Monarch Life Assur. Co., Winnipeg, Canada	F. A. L. Mathewson, M. D
Monarch Life Insurance Co., Springfield, Mass.	J. M. Gilchrist, M. D. L. E. Hathaway, Jr., M. D.
Montreal Life Insurance Co., Montreal, Canada	R. V. Ward, M. D.
Monumental Life Insurance Co., Baltimore, Md.	F. H. Vinup, M. D.
Mutual Benefit Life Insurance Co., Newark, N. J.	J. R. Beard, M. D. E. C. Hillman, Jr., M. D. W. A. Reiter, M. D. D. F. Steuart, M. D. Murray Wagman, M. D. Jefferson Weed, M. D.
Mutual Life Assur. Co. of Canada, Waterloo, Can.	J. M. Livingston, M. D. R. W. Zinkann, M. D.
Mutual Life Ins. Co. of New York, New York City	H. A. Bancel, M. D. W. M. Bradshaw, M. D. F. H. Carber, M. D. F. V. Costello, M. D. J. R. Gudger, M. D. J. F. Moore, M. D. S. A. Narins, M. D. E. J. Quinn, M. D. F. A. Waldron, M. D. R. L. Willis, M. D.
Mutual Savings Life Insurance Co., St. Louis, Mo.	A. C. Henske, M. D.
Mutual Trust Life Insurance Co., Chicago, Ill.	A. A. Willander, M. D.

National Fidelity Life Insurance Co., Kansas City, Mo.	J. V. Bell, M. D.
National Guardian Life Insurance Co., Madison, Wis.	A. R. Tormey, M. D.
National Life & Accident Ins. Co., Nashville, Tenn.	B. F. Byrd, M. D. G. E. Fort, M. D. L. C. Miller, M. D.
National Life Assurance Co. of Canada, Toronto, Canada	E. C. Noble, M. D.
National Life Co., Des Moines, Iowa	L. K. Meredith, M. D.
National Life Insurance Co., Montpelier, Vt.	G. E. Allen, M. D. H. L. Colombo, M. D. A. J. Oberlander, M. D.
National Old Line Insurance Co., Little Rock, Ark.	R. E. McLochlin, M. D.
New England Mutual Life Ins. Co., Boston, Mass.	F. R. Brown, M. D. M. H. Clifford, M. D. H. M. Frost, M. D. O. C. Hendrix, M. D.
New York Life Insurance Co., New York City	D. R. Auten, M. D. William Bolt, M. D. E. J. Campbell, M. D. G. D. Dorman, M. D. A. H. Faber, M. D. E. M. Freeland, M. D. E. E. Getman, M. D. W. D. Heaton, M. D. I. C. Lawler, M. D. R. W. Pratt, M. D.
North American Accident Insurance Co., Baltimore, Md.	Eduard Novak, M. D.
North American Life Assur. Co., Toronto, Canada	J. G. Falconer, M. D. Eugene Montgomery, M. D.

North American Reassurance Co., New York City	J. T. Montgomery, M. D.
Northern Life Assurance Co. of Canada, London, Canada	J. H. Geddes, M. D.
Northwestern Mutual Life Ins. Co., Milwaukee, Wis.	R. W. Benton, M. D. J. A. End, M. D. R. T. Gilchrist, M. D. W. G. Hyde, M. D. D. F. Rikkers, M. D. G. F. Tegtmeyer, M. D. D. E. W. Wenstrand, M. D.
Northwestern National Life Ins. Co., Minneapolis, Minn.	K. W. Anderson, M. D.
Occidental Life Insurance Co., Raleigh, N. C.	V. S. Caviness, M. D.
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Ohio State Life Insurance Co., Columbus, Ohio	T. F. Ross, M. D.
Old Line Life Insurance Co. of America, Milwaukee. Wis.	H. M. Hawkins, M. D.
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Peoples Life Insurance Co., Washington, D. C.	J. R. Biggs, M. D.
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Phoenix Mutual Life Ins. Co., Hartford, Conn.	H. B. Campbell, M. D. R. A. Goodell, M. D. Llewellyn Hall, M. D. N. R. Kelley, M. D. R. L. Rowley, M. D.
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Postal Life Insurance Co., New York, N. Y.	L. B. Dunn, M. D.
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Preferred Life Assurance Society, Montgomery, Ala.	B. C. Bird, M. D.
Protected Home Circle, Sharon, Pa.	W. G. McLaughry, M. D.
Protective Life Insurance Co., Birmingham, Ala.	E. G. Givhan, Jr., M. D.
Provident Life and Accident Ins. Co., Chattanooga, Tenn.	C. R. Henry, M. D.

Provident Mutual Life Ins. Co., Philadelphia, Pa.	E. J. Dewees, M. D. P. H. Langner, Jr., M. D. L. L. McLellan, M. D. D. S. Pepper, M. D.
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Western Reserve Life Insurance Company, Austin, Texas	C. P. Hardwicke, M. D.
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Wisconsin National Life Insurance Company, Oshkosh, Wis.	E. B. Williams, M. D.
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Subject Index

Vol. XXXI—1947

Note—The Names of Authors are Printed in Plain Type; of Discussers, in *Italics.*

Author Index

Vol. XXXI—1947

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